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NONARTERIAL DISORDERS SIMULATING DISEASE OF THE PERIPHERAL ARTERIES*

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THE clinician who suspects the presence of arterial disease in a patient's limb can usually make a positive diagnosis of such involvement. Occasionally, however, a disorder in some distant structure is responsible for the presenting symptoms, either through a direct action on the blood vessels of the extremity, or entirely independently of them. It is the purpose of this paper to call attention to the more frequent types of such distant lesions that cause difficulty in the differential diagnosis.

DISORDERS SIMULATING CHRONIC ARTERIAL DISEASE

The chief chronic organic disorders of peripheral arteries are arteriosclerosis and thromboangiitis obliterans, whereas the vasospastic disorder known as "Raynaud's disease" is the most important functional derangement.

The diagnosis of organic arterial disease depends considerably on the evidence of the diminution in arterial blood flow, which has been summarized[1] as follows: muscle action is weak, and intermittent claudication may develop; the limb is abnormally cold; hypesthesia, hyperesthesia and spontaneous pain may be present; there is diminished resistance to injury, and ulceration may result from slight trauma; the toes and feet show blanching on elevation, and rubor on dependency; and the pulsation in the affected arteries is either slight or absent.

Raynaud's disease is characterized by spasmodic attacks of coldness, with blanching or cyanosis in the fingers and toes, on exposure to cold. The major vessels are not affected, and the major pulses remain normal. The etiology of this infirmity is uncertain, some authorities attributing it to hyperactivity of the vasomotor system, others to a local vessel fault; but it will be here classified as a vascular disorder, from which other cases of vasospasm will be differentiated.

A great variety of conditions may resemble true arterial disease.

Arthritis

The pain of spinal arthritis commonly radiates down the upper and lower extremities. Von Bechterew,[2] in 1899, pointed out that the pain of arthritis may be due to the pressure of inflammatory exudate on the spinal nerves or their roots. In 1916, Nathan[3] demonstrated, in experimentally produced arthritis, thickening of the vertebral bodies pressing on the spinal cord, and the infiltration of exudate in and about the vertebral articulations encroaching on the nerve roots or trunks in the intervertebral foramina. It is probably justifiable to assume that such lesions occur often in arthritic patients.[4, 5] This thesis is further strengthened by the amelioration of neuritic symptoms after the application of spinal traction.[6]

In arthritis of the cervical spine, the neuritic symptoms may resemble those of Raynaud's disease or of cervical-rib disease.[7] Tingling of the fingers, often accompanied by hypesthesia and pallor, is apt to be the predominating symptom. In addition, poorly localized pain is usually present in and about the shoulder and in the entire extremity. These symptoms are often intensified by placing the limb in certain positions, which vary with the patient. An outstanding complaint is that the symptoms increase with recumbency and are intensified when the patient awakes in the morning.

Vascular disease may be further suspected because of coldness of the fingers and actual reduction in pulse volume (Fig. 1). The cervical nerves receive their sympathetic fibers beyond the inter-

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vertebral foramina; therefore, direct pressure on them or their roots cannot account for the vasospasm, but it may be a reflex from the joint irritation (Fig. 2). Dr. Thomas H. Peterson, in a personal communication, states that in such cases the application of head traction may cause a fall in the diastolic arterial pressure in the limb, with a corresponding increase in the amplitude of the pulse.

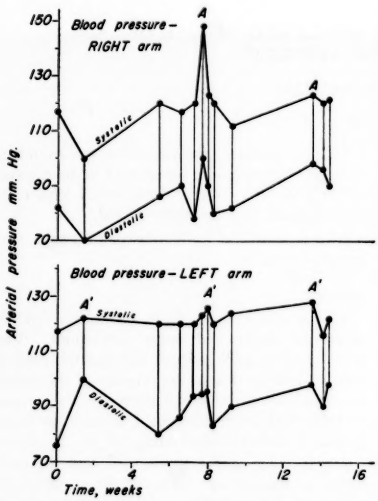

FIGURE 1. *The Brachial Blood Pressure in a Patient with Cervical Arthritis. (Courtesy of Dr. Thomas H. Peterson.)*

Pain radiating into the right upper limb occurred at A, *and in the left limb at* A'. *The blood pressure was high on the affected side during the attacks of pain, presumably because of vasoconstriction in the distal parts of the limb.*

Symptoms are not always present in the neck. Likewise, clinical examination may not reveal any abnormality there. Thus Turner and Oppenheimer,[7] in their reported group of 50 patients, most of whom, at least, were arthritic, found only 2 who complained of pain in the neck, and only 2 who had restriction of cervical motions. The history is nevertheless of extreme importance, inasmuch as the patients do not suffer most on exposure to cold air but, contrariwise, may be most uncomfortable in bed when the extremities are adequately warmed. The joints of the fingers may or may not show arthritic changes. The finding of either atrophic or hypertrophic changes in the cervical spine in the roentgenogram is, of course, significant.

CASE 1. A. T., a 40-year-old married woman, was referred for disagreeable prickling sensations in the fingers of both hands, associated with coldness and sweating. The onset was gradual in the early autumn and had lasted for 1 month prior to examination. The diagnosis of Raynaud's disease was considered, especially since two of the patient's sisters had disorders of this type. Further questioning elicited the fact that the prickling was present during the night, and was particularly troublesome when the patient awoke in the morning. She recalled no changes in the color of the hands. Likewise, there was no association of the attacks with exposure to hot or cold air or water.

Examination showed cold sweaty hands and fingers. There was no discoloration, deformity or trophic change, and sensation was grossly normal. Both radial pulses could be easily palpated, but the Pachon oscillometer showed a slightly diminished pulse expansion at the wrists, consistent with some vasoconstriction. Motion of the neck was full in all directions. Roentgenograms of the cervical spine showed a hypertrophic arthritis, with calcification of the intervertebral ligaments.

The patient received immediate benefit from diathermy treatment of the cervical spine. Recurrence of symptoms was expected, however, with any reactivation of the arthritis.

If arthritis attacks the lumbar spine, symptoms are experienced in the lower limbs, analogous to those described above. The pain does not often lie along the sciatic nerve, but is apter to be found in other regions of the limb. In the foot, it may be accompanied by hypesthesia and paresthesia, an association strongly suggestive of arterial insufficiency. More typical, however, is poorly localized pain in the thighs. This is ordinarily not diminished on activity, and occasionally its exacerbation on walking may be associated with cramps, which differ from those of true intermittent claudication in their location and lack of regularity. As in the upper extremity, vasoconstriction may be marked. It is noteworthy that the symptoms are by no means necessarily bilateral.

CASE 2. S. G., a 62-year-old tailor, complained of coldness and pain in the left lower leg. Fourteen years previously, he had an attack of severe pain in the left foot and ankle, necessitating bedrest for several weeks. Three years before, he had a recurrence of the pain, which disappeared after massage. Three months before the patient was seen, the pain again returned. It was described as a "drilling" or "pressing" in the left foot and ankle, and was initiated by standing or walking. Walking was indeed almost impossible, since the pain forced the patient to stop for a few minutes' rest about every 25 feet. The discomfort disappeared immediately on resting. He also described an additional, more constant pain throughout the thigh, which was increased on recumbency. The patient refused to stay in a supine position on the examining table, because it induced cramps in his left thigh. It was brought out that these cramps frequently occurred at night.

Examination showed coldness of both feet, with the surface temperature of the left foot 3°C. below that of the right. There was no blanching on elevation, and no rubor on dependency. All the arterial pulsations were palpable, with the exception of that of the left posterior tibial artery. The Pachon oscillometer showed a diminished pulse amplitude, the reading of the right ankle being 2.5 units, that of the left 2.0 units. The spine showed a loss of the normal anterior convexity of the lumbar region, with an extreme right dorsal, left lumbar scoliosis, in severity; back exercises were instituted. After the ninth treatment, he felt greatly relieved, and diathermy was discontinued.

If the arthritis involves the joints of the limb, it is usually a simple matter to avoid its confusion with vascular disease. In cases of pain in the foot, for example, immobility on manipulation may point to involvement of the small joints of this

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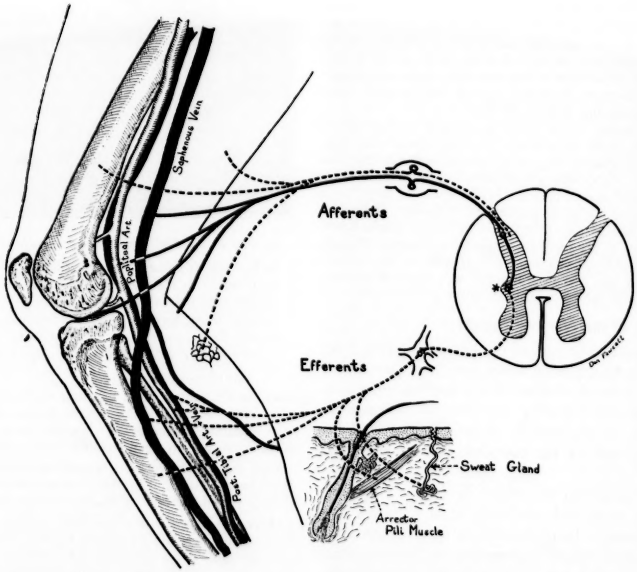

FIGURE 2. *Pathways for Vascular Reflexes Originating in the Limb (modified after de Takats[17]).*

Afferent impulses can arise in any of the tissues, including bone, joint, skin, artery and vein. The stimuli travel by way of the sensory fibers of the spinal nerves (solid line) to reach the cells of the intermediolateral column of the cord. These cells (*) represent a "final common path" of the sympathetic system, since they receive impulses from all levels of the nervous system, and send their processes outward to the sympathetic ganglia. Postganglionic fibers conduct the efferent impulses to the arteries and veins (constriction), as well as to the sweat glands and arrector pili muscles. Some sensory fibers of the spinal nerves (broken line) conduct afferent impulses from the skin or deep tissues and, through an axon reflex, give rise to the efferent response of vasodilatation in the capillaries of the extremity.

but good flexibility. X-ray examination showed prominent spurs at the margins of the lower dorsal and lumbar vertebrae, with calcific bridging of the 1st lumbar intervertebral space. The hip joint likewise showed hypertrophic changes.

The patient was placed on a high-vitamin, low-carbohydrate diet, and diathermy treatment was given to his spine. The feet became warmer, and the pulses increased somewhat in amplitude. After four such applications, the patient could lie flat on his back without cramps in the thigh, and the symptoms on walking had diminished

part. Arthritis of the knee may cause pain radiating into the calf. Here one should recall that the pain of an arthritic knee is usually worse on arising, but diminishes with moderate use. Examination will show periarticular thickening, with or without crepitus and pain on motion.

Decision is difficult when the pain is increased by walking, particularly if it is cramplike. This is perhaps seen oftener when the arthritis involves

the hip. As in spinal arthritis, this cramp is differentiated from that of true intermittent claudication by its irregularity in time and in degree of severity. Clinical examination may not demonstrate disability in the hip joint, but involvement of the more accessible joints points to the diagnosis, and the roentgenogram may be of great aid.

Case 3. L. S., a 40-year-old housewife, was referred for pain in the lower extremities of 1 year's duration. The pain appeared in both ankles while the patient was walking, but it was less troublesome there than in the upper medial part of the left thigh, where it was cramp-like. The patient had to stop and rest because of the pain, but the distance traversed between rest periods varied from time to time and day to day.

Examination of the lower extremities showed strong pulsations in all the arteries. The saphenous veins were moderately dilated, and some valvular incompetence was manifest. There was a little loss of mobility in the feet, but no evidence of arthritis in the knees or back, or in the joints of the upper extremities. Extremes of hip motion reproduced the crampy pain in the left thigh. Roentgenograms of the hip joint showed hypertrophic changes. The patient's symptoms were attributed to the arthritis of the left hip, and improvement was obtained by treatment similar to that mentioned in the previous case.

Diseases of the Nervous System

Lesions of any part of the nervous system, from the brain to the finer nerve branches, may mimic chronic arterial disease. As a well-known example, one may mention the cold and cyanotic extremities in the chronic stage of anterior poliomyelitis, and in the catatonic type of dementia praecox.

The simulation to arterial disease is frequently brought about independently of any known vascular change. In these cases, it may be especially confusing if trophic ulceration is present, since ulceration often has its origin in intrinsic vascular disease. Medical writers have unfortunately caused some confusion by using the term "mal perforans" for any perforating lesion of the foot or toe, regardless of the etiology. The presence or absence of sensitivity in such a lesion is, happily, an excellent guide to its true etiology. When the ulcer is of vascular origin, there is usually severe pain. The trophic ulcer of nervous origin, on the contrary, is usually located in a zone of hypesthesia or anesthesia. A determination of the neurologic state of the limb and the patient, on the one hand, and the vascular state, on the other, allows one to establish the correct diagnosis.

Case 4. M. M. (Boston City Hospital No. 79470), a 39-year-old laborer, was first seen at the outpatient department of the hospital for a septic callus over the plantar surface of the left 5th metatarsal. Amputation of the 5th toe and head of the metatarsal was performed because of the presence of osteomyelitis.

Six months later, the patient developed an ulcer on the plantar surface of the left great toe (Fig. 3). The foot was cold, sweaty and cyanotic, findings that suggested an underlying vascular lesion; both the dorsalis pedis and posterior tibial pulses were normal, however. The plantar surface of the toe, including the base of the ulcer, was so anesthetic that the underlying bone, which was carious, could be probed without producing the slightest pain.

Further questioning revealed that the patient had fallen down the hold of a ship and injured the spine, 12 years previously. He was treated by a plaster cast, then by a brace, for 2½ years. From that time on, he had no symptoms until the onset of the present illness. He admitted some dribbling of urine for 3 weeks prior to the last examination, but no rectal incontinence and no impotence.

Figure 3. *Mal Perforans of the Great Toe.*

In this patient (Case 4), the condition was associated with injury to the lumbar nerve roots following a compression fracture of the spine, but the peripheral arteries were normal. Carious bone was present in the floor of the ulcer. The fifth toe had previously been amputated for the same disorder.

X-ray examination showed an old compression fracture of the body of the 2nd lumbar vertebra, with bony union between it and the body of the 1st lumbar vertebra.

There was hypesthesia of the body on both sides below the distribution of the 1st lumbar nerves. The knee jerks were diminished on the right side, and the ankle jerks were absent. There was a positive Babinski reaction on the left side. A laminectomy was performed by Dr. Walter Wegner. There was demonstrated a considerable mass of scar tissue, both fibrous and bony, at the level of the 1st and 2nd lumbar vertebrae, involving some of the anterior and posterior roots of the cauda equina. The scar tissue was removed, some of the roots being unavoidably injured during this procedure. The symptoms gradually regressed after operation, and the ulceration of the foot healed after several months.

Peripheral neuritis of various forms may simulate arterial disease even when unaccompanied by ulceration. This is due in part to the presence of pain in the limbs, and in part to coldness incident to vasospasm. In this connection, Lewis and Pickering[8] state:

> The chief cause of lowered skin temperature after mixed nerve lesions is loss of the fibers belonging to the posterior root system. While it is probable that this loss is effected chiefly through the disappearance of the sensory axon reflex [normal vasodilatation mechanism], we are unable to exclude the possibility

that loss of vasodilator impulses passing from the cord by posterior root fibers may also play a part. (See Fig. 2.)

Dole and Morison,[9] however, could find no evidence for the existence of the second mechanism mentioned.

CASE 5. L. J., a 42-year-old jeweler, was referred for cramps in the feet and calves of 8 years' duration. The cramps came mostly at the close of any day that required much standing. On some occasions, they appeared while the patient was standing and could be relieved by walking, whereas at other times, they came on only after traversing a block or two. For 3 years before consultation, the patient was troubled by this symptom at night, which necessitated his getting out of bed for a few seconds, when the cramps disappeared. The patient's malady was worse in the 3 weeks prior to the examination, a fact attributed to a temporary increase in work. Additional evidence was secured that the patient had been a fairly hard drinker for many years. During the month preceding examination, he had taken several drinks a day and finally had been confined to his home for several days because of alcoholism. He had become extremely apprehensive and had entertained some delusions. His diet was admittedly poor, with a minimum of fresh vegetables. He had begun smoking at the age of 16 and, at the time of examination, smoked ten to fifteen small cigars a day. Chronic otitis media and mastoiditis were present and possibly operated as additional toxic factors.

Examination showed moist, cold feet. There was no discoloration and no blanching on elevation. The femoral and popliteal pulses were excellent. Although neither dorsalis-pedis pulsation could be felt, the posterior tibial pulses were of excellent quality. The oscillometer showed small but equal pulse expansions at the ankles. There was some superficial tenderness on the dorsum of the feet, and a little extensor weakness. Both feet were in moderate pronation.

It was believed that the coldness and sweating represented vasomotor symptoms due to an alcoholic neuritis or, possibly, to the patient's smoking. Relief from the cramps was obtained by an increase in vitamin intake, a reduction in smoking and measures directed to pronation of the feet, namely, the use of orthopedic heels and foot exercises.

So-called "ganglionitis" and "radiculitis" are troublesome variants of peripheral neuritis.[4, 5] The presenting symptom usually is intractable pain in the foot or leg, which on close questioning is characteristic of a hyperesthesia. The symptoms vary according to whether the posterior or anterior roots, or both, are involved. Examination may disclose areas of hyperesthesia or anesthesia, with muscle weakness and abnormal deep reflexes. The majority of cases occur in the lower extremity.

CASE 6. C. H., a 57-year-old housewife, was referred because of pain and coldness of the right foot, and cramps of the right calf, of several years' duration. There was fairly constant pain and blanching in the 2nd toe. The remainder of the foot was cold and blanched spasmodically, especially in cold weather. The patient avoided walking, because of heaviness in the leg. This symptom was usually not accompanied by cramps, which were apter to appear at night. The patient believed that at least some of her symptoms dated back 39 years to an attack of herpes zoster that had involved the right lumbar region.

Examination of the lower limbs showed normal cutaneous color in all positions. Both feet were cold, however, the right more than the left. The right dorsalis-pedis pulse could not be felt, but its absence was apparently adequately compensated for by a palpable peroneal and a large posterior tibial pulse. The left dorsalis-pedis and posterior tibial pulses were forceful. Oscillometric examination showed good arterial pressure and pulse amplitude at both ankles.

In contrast to its normal vascular status, the limb showed abnormal neurologic findings. The patient was transferred to the care of Dr. Tracy J. Putnam, whose findings were as follows:

> Motor power was good in both legs and both feet; the right ankle jerk was more active than the left, however, and there was a suggestion of clonus on the right. The Babinski reaction was negative on both sides. There was hyperesthesia over an area of the dorsum of the right foot centering around the 2nd toe and plantar surface of the 2nd and 3rd toes. The hyperesthesia was most marked to pinprick. There was a loss of vibratory sense in the ankle, but no loss of position sense. There was no tenderness of the sciatic nerve, but there was tenderness of the lower spine. Small scars in the mid-line were suggestive of an old herpetic eruption.
>
> It seems to me that the diagnosis must be a chronic progressive neuritis. In view of the old history of pain, the long-continued subjective heaviness of the right leg and the hyperesthesia, this must have affected the whole sciatic nerve to some extent, but is most intense in the peroneal branch. I believe that it is a result of the old herpes.

According to Dr. Putnam's suggestion, the patient was given deep x-ray therapy over the roots of the sciatic nerve. After ten treatments, she experienced complete relief from her symptoms.

A more puzzling interplay of neurologic and vascular symptoms is present in the syndrome of causalgia, resulting from injuries to the large nerves, especially the median and sciatic. The symptoms, which are disagreeable and disabling, consist in extreme hyperesthesia, with coldness, cyanosis and sweating.[10–12] Similar disorders of less severe degree may follow almost any injury in any part of the extremity. Such cases following a fracture, sprain, puncture wound or mild infection have been described by several authors.[13–18] As emphasized by de Takats,[17] this category of reflex dystrophy of the extremities includes syndromes designated as reflex vasoconstriction, traumatic osteoporosis, traumatic arthritis and so forth. Common to all such disorders is an irritation of a sensory nerve or its endings, which sets up reflexes mediated by the sympathetic fibers with an effector response in the blood vessels (Fig. 2). The sweat glands and arrector pili muscles may also react, thus giving rise to locally

excessive sweating and to an exaggerated pilomotor ("goose-flesh") reaction. Homans[18] adds a theory that some of the sensory changes are due to the interference with the nutrition of the larger nerve trunks caused by the reflex vasoconstriction of their nutrient arteries.

When a diminution in blood flow results from such disorders, it becomes necessary to rule out organic obstruction of the arteries, which indeed may have been produced by the primary injury to the limb. The decisive data are obtained by

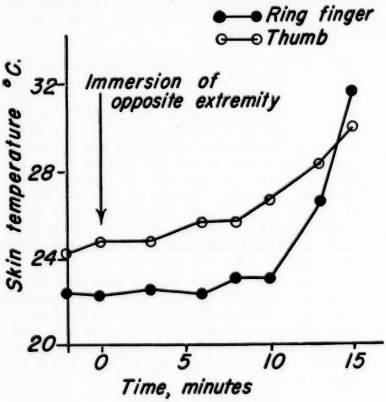

FIGURE 4. *A Vasodilatation Test in a Patient (Case 7) with Vasospasm Secondary to a Scar Neuroma of the Ring Finger.*

Immersion of the opposite extremity in warm water resulted in a pronounced vasodilatation of the affected finger and a rise in the skin temperature to a level at least as high as that of the normal first digit. The test established that the diminution in blood flow was reflex in nature and ruled out organic disease of the peripheral arteries.

inhibiting the vasomotor control of the vessels.[19] The uninvolved extremities may be immersed in hot water at 43°C. (Landis–Gibbon test), or spinal anesthesia may be induced for the lower extremities and paravertebral sympathetic anesthesia for the upper extremities. If the diminution in blood flow depends entirely on vasospasm, without any organic vascular obstruction, the sympathetic inhibition allows a considerable increase in blood flow to occur. This effect can be measured by determining the skin temperature or the oscillometric index of pulse expansion, before and after the test (Fig. 4).

When one is dealing with a reflex effect, relief will be obtained if the irritable focus can be excised, as in the case described below, or if the reflex can be interrupted, as by sympathetic ganglionectomy.

CASE 7. M. A. H., a 29-year-old factory worker, was seen for pain and coldness of the right ring finger. Seven months prior to consultation, a sliver of steel had passed under the nail, and the finger had become infected. The finger tip was incised under procaine block at the base of the digit, and under general anesthesia on two more occasions. Immediately after the last incision, the finger became white, cold and painful. The pain was constant, necessitating sedation at night. The slightest touch of any part of the finger resulted in intense pain, and the symptoms were likewise increased on exposure of the hand to cold. The patient was not working. He was depressed mentally, and had lost 18 pounds.

Examination showed the finger to be blanched from base to tip. Both this digit and the little finger were strikingly cold, the temperature being 2.5°C. below that of the other digits. An exquisitely tender scar, 8 mm. long, was present on the tip of the injured finger, but the hyperesthesia extended beyond this zone onto both surfaces of the finger to its base. The pulses at the wrist were normal to palpation and by oscillometric measurement.

The obvious marked diminution in local blood flow brought up the possibility that the digital arteries had been thrombosed during the infection, or by the digital nerve block. To determine whether the vessels were thus obstructed, the cutaneous temperature of the fingers of the right hand was measured before and after immersion of the normal left hand and forearm in hot water. Adequate vasodilatation resulted (Fig. 4). The digital arteries were therefore not organically blocked but, on the contrary, were in a state of vasoconstriction, apparently secondary to the tender scar of the finger tip.

Excision of the scar resulted in the complete disappearance of the disorder.

Phlebitis

It is known that phlebitis is often accompanied by vasospasm, manifest by small pulses and coldness in the extremity, and may thus resemble a primary arterial disorder.[20] It is quite evident that, from the standpoint of the mechanism of this vasospasm, phlebitis is, like the other conditions considered above, one particular source of sensory irritation that is adequate to set off vasoconstrictor responses.

In a previous report,[20] it was emphasized that the arteries are but one of several structures in the limb that, because they receive sympathetic innervation, are all equally liable to react to venous irritation. These structures include the veins, the sweat glands and the arrector pili muscles (Fig. 2). Therefore, if arteriospasm is present, it may be associated with sweating of the cold extremity and an abnormally active pilomotor reflex ("goose-flesh").

Severe arteriospasm giving loss of the major pulses may occur, but is much less common than minor degrees of vasoconstriction. To establish

the true nature of the disturbance, one must look for the typical tender induration of the phlebitic vein, and the cyanosis and edema on standing. Phlebitis may further be suspected as the cause of the diminished arterial flow if there are accompanying sweating and increased pilomotor response. The latter reflex is easily tested by pinching the skin of the extremity, or by touching it with a cold object. Finally, one should be able to demonstrate that the blood flow can be increased, after sympathetic release, by one of the methods described in the preceding section.

CASE 8. M. P., a 40-year-old married schoolteacher, was referred for pain and coldness of the right foot, following a fracture of the 5th metatarsal bone. These symptoms were constant, and increased on standing or walking, when they were accompanied by cyanosis and edema of the limb. For some months prior to the accident, the patient had noticed cramps, which appeared during the night but not while she was walking. With this history in mind, and because no pulsations could be made out in the pedal arteries, her local doctor had suspected arterial disease and had treated the patient by injections of a pancreatic extract (Depropanex). At the end of 2 weeks' bedrest, the swelling diminished but the extremity was still painful and cold.

On examination, the right calf showed 1.3 cm. of atrophy. There were moderate cyanosis and coldness in the lower leg, foot and toes. The toes blanched slightly on elevation. There was no excessive sweating, but the pilomotor response was exaggerated. The femoral and popliteal pulses were of good quality, and although the pulsation of the dorsalis-pedis artery could not be felt, that of the posterior tibial artery was fair. On the unaffected left side, the pulsations were approximately the same, with a nonpalpable dorsalis-pedis but a slightly larger posterior tibial pulse. Moderate edema was present on the medial side of the affected ankle, and a tender thickening could be felt along 12 cm. of the course of the saphenous vein in the lower calf. There was but minimal tenderness over the 5th metatarsal bone, and an x-ray film showed good healing of the fracture.

The nocturnal cramps preceding the injury were presumed to be due to a varicose widening of the saphenous vein. This was borne out by the discovery of a dilated vein at operation. The tenderness over a segment of the vein, in addition to the cyanosis and edema, pointed to a phlebitis that might have started in the deep veins following the primary trauma, with subsequent extension to the saphenous vein. The coldness and exaggerated pilomotor response were consistent with a sympathetic reaction from the phlebitic vein.

A ligation of the saphenous vein was performed under spinal anesthesia. Skin temperature and oscillometric readings showed adequate vasodilatation after the anesthesia and operation, a finding indicating that there was no organic obstruction of the arteries. Conversely, the experiment strengthened the conclusion that the coldness of the extremity was due to a vasospasm, presumably secondary to the phlebitis.

The symptoms quickly diminished. Small areas of thrombophlebitis appeared in the superficial veins of the thigh. Radiant heat and diathermy were given to the limb, and active exercises were started. On discharge, the same treatment was continued by the family physician, and the symptoms regressed satisfactorily.

Cervical Rib, Anomalies of the First Thoracic Rib and the Scalenus Syndrome

Although cervical rib is usually included in a classification of vascular disease, it is more properly considered as a condition that simulates primary arterial disease.

The symptoms can be both neurologic[21] and vascular, and may be constant or spasmodic. The patient usually complains of numbness and tingling in the limb, especially along the medial side of the forearm and hand and in the more medial fingers. Muscular weakness and wasting may occur in the hypothenar muscles or, less commonly, in the thenar group. True vascular symptoms are infrequent. They include episodes of blanching or cyanosis of one or more fingers or of the entire hand. Both sets of symptoms occur, especially in relation to muscular effort or to changes in position of the neck or the extremity. Exposure to cold may inconstantly initiate an attack. The blood pressure may be lowered on the affected side, and the radial pulse may be small and, very rarely, obliterated. Pain may be felt in the neck, radiating into the shoulder. If the rib is large, there may be a visible elevation. In some cases, the subclavian pulsation is more prominent than normal; this happens most frequently when the rudimentary rib belongs to the first thoracic segment.[22]

It is apparent that the neurologic symptoms are caused by the impingement of the lower trunk of the brachial plexus on the cervical rib[21, 23, 24] (Fig. 5). Todd[25] and others[26, 27] believe that vascular symptoms, when present, are caused by the concomitant irritation of the sympathetic fibers in the trunk, whereas Lewis and Pickering[28] and others[29] conclude that these symptoms depend on actual contact of the rib and the subclavian artery. Murphy[30] suggested that the scalenus anterior muscle, placed in front of the artery and nerve, plays a role in the production of symptoms by pressing these structures against the rib. This thesis was reaffirmed by Adson and Coffey.[31]

It is worth remembering that an abnormal first thoracic rib may give rise to symptoms identical with those of a cervical rib.[23, 32, 33] Finally, there is a much larger group of patients, with the signs of cervical rib, who show no bony anomalies.[34–37] Here, as in cervical rib, symptoms are apparently caused by injury to the lower trunk of the brachial plexus as its component from the first or first and second thoracic nerves arches upward and outward over the first rib to gain the limb (Fig. 5). An exaggeration of the downward slope of the shoulder (especially pronounced in women) may initiate, or at least aggravate, this irritation. Naff-

ziger and Grant[87] have emphasized the role of the scalenus anterior muscle in forcing the trunk backward against the rib, and have applied the name of "scalenus syndrome" to these cases.

The diagnosis of cervical rib should be seriously considered when an apparent vascular disorder is present in a single upper extremity, without symptoms in the lower limbs. It differs from Raynaud's disease, first, in its usual lack of symmetry and, again, in that attacks of pain and vascular disturbance are inconstantly precipitated by exposure to cold. It is apparent, from the consideration of the mechanism of symptoms, that the typical effects of either cervical rib or the scalenus syndrome can be elicited by any procedure that increases the pressure of the rib against the brachial plexus or the subclavian artery, or both. The usual recommendation is for the examiner to pull the arm forcibly downward, while the patient bends the head sharply to the opposite side, but with the chin tilted toward the involved side (scalenus maneuver). The test is more effective if the patient simultaneously takes a deep breath, thus elevating the uppermost rib against the trunk and artery.

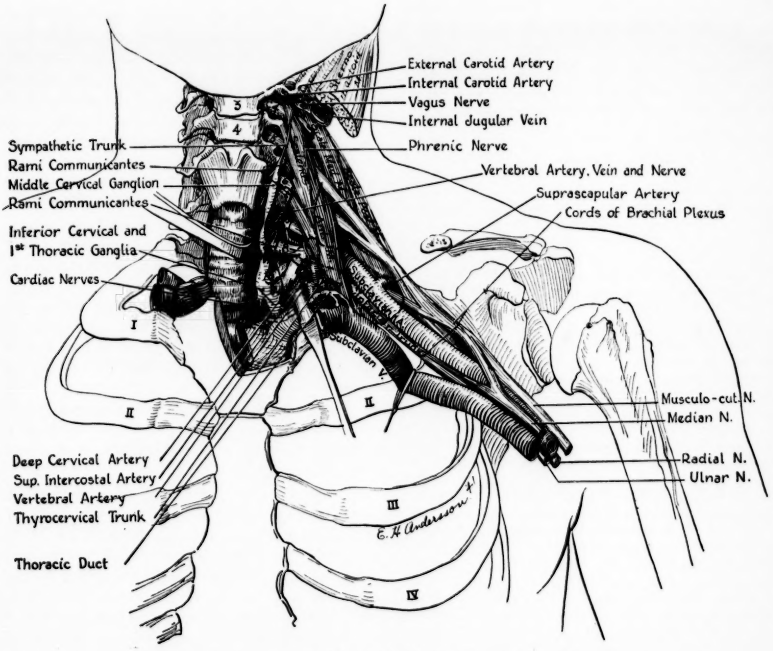

FIGURE 5. *Structures Involved in the Production of the Scalenus Syndrome.*

The dissection shows the roots of the brachial plexus and their communications from the sympathetic ganglia. Note the scalenus anterior muscle thrusting the lower trunk of the brachial plexus and the subclavian artery against the first rib. The apex of the pleura and the first part of the subclavian artery are depressed by the retractor. The thyroid cartilage lies slightly lower than usual.

CASE 9. E. F., a 57-year-old housewife, complained of numbness and weakness in the left upper arm, with tingling in the fingers, of about 15 years' duration.

Forceful motion of the arm precipitated attacks of numbness in the medial side of the left forearm, extending through the entire hand; tingling was simultaneously present in the fingers. Sudden muscular weakness forced the patient to drop any object that she held in the hand. The symptoms also arose if she sat back in a chair in a relaxed state. She obtained relief by sitting forward, hunching the shoulders and massaging the fingers.

On examination, the radial and ulnar pulses were normal. There was no muscle atrophy, and no demonstrable change in sensation. A knoblike structure could be seen and felt in the middle of the left posterior triangle of the neck. The patient had not complained of discomfort on the right side, and no abnormality could be palpated there. Nevertheless, the scalenus maneuver caused the right radial pulse to disappear, and the brachial blood pressure to fall to zero. On the left side, the maneuver brought on

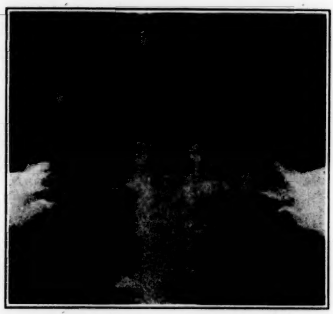

FIGURE 6. *Roentgenogram of Bilateral Cervical Ribs.*

In this patient (Case 9), the right rib is of moderate length, ending in the posterior cervical triangle. The left extends forward to articulate with a process springing from the first thoracic rib.

such a disagreeable attack of numbness and tingling that the patient did not permit further observations. A roentgenogram made by Dr. Paul F. Butler showed a short cervical rib on the right side and a long cervical rib on the left (Fig. 6).

Operation was advised, but refused.

Solar Irradiation

A startling picture of edema and mottled cyanosis of an extremity may be produced by exposure to sunlight. Such an appearance, continuing for some weeks, is seemingly due to a long-lasting dilatation of the veins of the part, an effect of irradiation that was revealed in a former study.[38] The condition may be confused with phlebitis, and occasionally, as in the following case, thromboangiitis obliterans with deep venous involvement may be suspected.

CASE 10. A. McL., a 47-year-old janitor, was referred for a "blue-red" discoloration of the legs and coldness of the feet.

The disorder was said to have started after a severe sunburn 4 weeks previously. The day following the exposure, he returned to work but was forced to leave after 2 days, because of painful swelling of the ankles and shins. Bed-rest relieved the swelling, but the cyanosis of the legs and the coldness of the feet remained. Thromboangiitis obliterans had been considered a possible diagnosis.

Examination showed prominent cyanosis of the shins and dorsums of the feet. This sign increased in intensity when the patient stood. There was no edema, and no induration or tenderness of the veins. The feet were cold but did not blanch on elevation. Both dorsalis-pedis and posterior tibial pulses were palpable, and the oscillometer showed a satisfactory pulsation at each ankle.

The symptoms were attributed to a venous dilatation caused by the exposure to sunlight, and a spontaneous cure was prognosticated. The patient was advised to return to work. Recovery was slow but complete.

DISORDERS SIMULATING ACUTE THROMBOSIS OR EMBOLISM

The diagnosis of acute arterial obstruction is unmistakable when there is sudden pain over a large artery accompanied by absent pulsations and a blanched, palsied and hypesthetic limb. In rare cases, the obstruction is effected, not, as ordinarily, by thrombosis or embolism, but rather by a massive arteriospasm. The rarity of such a phenomenon is noteworthy. Thrombophlebitis has been the usual causative factor in reported cases; however, I have seen one patient in whom a disorder of the central nervous system gave rise to a similar reaction.

Thrombophlebitis

At the inception of thrombophlebitis of the larger veins, a sudden severe spasm of the accompanying artery may occur. A report of such a reaction has been presented in a previous paper,[20] and the reader is referred to it for references to similar published cases. The reaction differs only in degree from the more gradually appearing and longer lasting arteriospasm already mentioned as simulating chronic arterial disease. It is noteworthy that, in all the recorded cases, the massive arterial spasm appeared at the onset of the phlebitis, rather than during the course of the malady.

The particularly confusing feature in these cases is the frequent absence of the typical symptoms of thrombophlebitis at the time when the vasospasm is noted. The color of the extremity after a moment's elevation may be of aid in arriving at the diagnosis, for a limb blanches quite completely when the artery alone is obstructed but retains much of its cyanotic tint if thrombophlebitis is present. As in the less severe reactions described earlier in this paper, other sympathetic reactions, particularly an exaggerated pilomotor response, may be present and may point to phlebitis. Finally, if one sees the patient an hour or more after the onset of pain, cyanosis is usually marked, dilated superficial veins appear, and the involved vein may be palpably indurated and tender.

The main features of the case previously reported[20] are as follows:

CASE 11. A 46-year-old man entered the hospital because of pulmonary embolism resulting from a phlebitis in the deep veins of the right leg. Within the next two weeks, the phlebitis ascended to the right femoral and iliac veins. This was followed by sudden pain in the left groin and inability to move the extremity. The left leg was cold, dry and anesthetic to the knee. Above, it was hyperesthetic. Before this episode, all the pulses of the leg had been palpated and found to be normal, but shortly thereafter no pulsation could be detected in any of the vessels from the femoral artery downward.

The symptoms disappeared and the pulsation returned in full force within 45 minutes after the application of heat.* Induration of the left femoral vein was then apparent, and dilated superficial veins were visible in the left flank.

Disease of the Central Nervous System

It has already been shown how diseases of the nervous system, both central and peripheral, may simulate chronic arterial disorders. The following case report is considered of particular interest, since an acute spasm of a major artery was induced by an intermittent disorder of the brain. It is possible that the cerebral cortex was involved in this reaction, since reported experiments indicate that the central and precentral areas of the frontal lobe are concerned in vasomotor control.[39] Peet and Kahn[40] report syndromes similar to Raynaud's disease that are incident to cerebral lesions, and refer as well to examples of the loss of a peripheral pulse during operative manipulation of the brain.

CASE 12.† J. J. (Boston City Hospital No. 800477), a 34-year-old woman, 19 days after a nephrolithotomy, suffered a sudden attack of transient numbness and weakness of the entire left side of the body, with a persistent right homonymous hemianiopsia. Because of a previous history of rheumatic heart disease, this incident was ascribed to a cerebral thrombosis or embolism. One week later, the patient was seized with a sudden knifelike pain in the left groin. The limb was numb, palsied and cold, and the resemblance to arterial embolism was further increased by the absence of the femoral pulse. Operative exploration revealed a tightly contracted femoral artery, the pulsation of which, however, returned when it was stripped from its sheath. No embolus was found on incising the artery, and following operation the pulses returned in the extremity.

I saw the patient through the kindness of Dr. Tracy J. Putnam, to whose service she was transferred. Determination of the vascular status of the lower extremities showed small but probably normal pulsations; those on the left side were only slightly weaker than those on the right. The variation was of a magnitude often seen in normal extremities.

During the remainder of her hospital stay, the patient suffered attacks of probable cardiac asthma associated with occasional incidents of numbness and weakness of the left side of the body. In some of these attacks, the pulses of the lower leg disappeared and the retinal arterioles became constricted. A similar attack, of greater severity, was induced by an intravenous injection of Ergobasine. Again the left femoral pulse disappeared, and the retinal vessels were seen to be constricted. The symptoms gradually wore off after the inhalation of carbon dioxide and oxygen.

Although certain aspects of the case pointed to the presence of hysteria, at least as a complicating factor, the consensus was that in the spontaneous episodes the patient had suffered from a cerebral and ophthalmic arteriospasm, which was associated with a spasm of the vessels of the left lower extremity. It was thought that this process had been repeated during the induced attack.

*If the condition had been true arterial embolism, local heating might have induced gangrene.

†This case is presented with the kind permission of Drs. I. J. Walker and S. J. G. Nowak, who reported it at a meeting of the Boston Surgical Society on February 3, 1936.

SUMMARY

Chronic disease of the peripheral arteries, as well as acute thrombosis and embolism, may be simulated by a great number of disorders in the other tissues of the extremities or in quite distant organs. The mechanisms whereby the confusing symptoms are produced are discussed, and several case reports are cited to illustrate the methods of differential diagnosis. These cases include arthritis of the cervical and lumbar spine and of the hip joint, a cauda-equina lesion from an old compression fracture of a lumbar vertebra, ganglionitis, peripheral neuritis, scar neuroma, phlebitis, cervical rib and the postirradiation state. Peripheral arterial embolism was suspected in a patient with phlebitis and in one with probable cerebral arteriospasm. Doubtless several other diseases could have been represented.

It is apparent that a precisely detailed history usually gives the clue to the correct diagnosis. One must also attend to the general examination and evaluate carefully the condition of all tissues of the limb. Thus, in addition to the condition of the vascular system, the examiner must determine the cutaneous, neurologic, articular, osseous and muscular status. If arterial inadequacy is shown to exist, he must discover whether it is due to organic disease or to vasospasm secondary to some other disorder.

23 Bay State Road

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WHAT SENSIBLE LIVING AND NATURAL RECOVERY CAN DO FOR A CARDIAC PATIENT*

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IN THESE days of high-pressure sales of medicines and the introduction of new surgical procedures, it is well to pause a bit and consider what may be accomplished by natural processes of healing and by sensible attention to the details of daily life. This observation applies to much of the field of internal medicine and especially to cardiac patients, most particularly those with rheumatic and coronary heart disease.

Much too often, recovery from a serious cardiac ailment is credited to a particular form of therapy, whether oral medication or surgical intervention. Follow-up studies have shown in many cases not only that recovery from an acute cardiac illness has taken place without special therapy, but also that these patients have lived useful lives for many years afterward. Such recovery and longevity, as will later be shown in illustrative case reports, occurs irrespective of the type of therapy instituted.

In a previous communication[1] attention was called to the fact that, although the prognosis of heart disease in apparently serious and grave cardiac states is often a difficult problem, one should recognize the fact that "functional recovery may be so complete that the ultimate prognosis is good for many years after."

Although one may encounter a patient with what appears to be a mild heart attack who unexpectedly dies, we have seen the reverse in many cases — that is, very seriously ill patients who not only make an immediate recovery but are able to carry on their occupations, with some limitations, for many years. The age of the patient and the type of cardiac disability may play little or no role in the ultimate recovery.

Because of the importance of these considerations and of the frequency with which they are overlooked, we have assembled the records of 7 striking cases illustrative of a great many that we have seen. The types of heart trouble were: rheumatic heart disease with mitral stenosis (2 cases) and coronary-artery disease (5 cases). We have divided the patients into two groups: those experiencing a natural, that is, spontaneous, recovery (3 cases) and those in whom longevity was due, in part at least, to sensible living (4 cases). The term "natural recovery" refers to a return to

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health that we could not ascribe to any particular type of therapy.

CASE 1. J. E. K. was first seen in the Cardiac Clinic at the Massachusetts General Hospital in June, 1923, at the age of 13, when rheumatic heart disease with active rheumatic fever was found. In November, when there was no evidence of active (rheumatic) infection, he indulged in football without any ill effects. In April, 1924, because of what was thought to be a well-marked mitral stenosis, he was referred to the Peter Bent Brigham Hospital for valvotomy. Because of the presence of asthmatic bronchitis, operation was deferred. The patient returned to the Cardiac Clinic in 1931 for a check-up; x-ray examination showed marked prominence in the region of the pulmonary conus, with a normal transverse diameter of the heart. The electrocardiogram showed sinus arrhythmia at a rate of 70, with notched P waves in all leads. The findings were consistent with rheumatic heart disease. On October 11, 1934, the patient returned feeling well and without symptoms, and on April 30, 1940, at the age of 30, he again returned to the clinic. Examination showed a healthy appearing man. The blood pressure was 120/75. The pulse rate was 60 and regular. There was no abnormal pulse in the neck. The heart was normal in size. With the patient in the upright position, the only abnormality heard with the Bowles stethoscope was a very slight early diastolic blow at the lower end of the sternum and at the apex; this murmur could easily have been missed. With the bell at the apex, there was a rather loud 1st sound, and a 3rd sound with a very faint rumble following it. With the patient in the recumbent position, the mitral diastolic murmur was louder, with slight presystolic accentuation.

In this case there was no doubt about the presence of a relatively slight amount of mitral stenosis and of a very slight amount of aortic regurgitation, neither of which is apparently a serious burden to the heart itself. The "marked mitral stenosis" noted sixteen years previously was probably largely due to dilatation of the heart secondary to acute rheumatic infection. This patient has done very well, and is lucky to have escaped operation. The case illustrates the extreme care that one must exercise in making a correct diagnosis in the first place, and the natural tendency for the heart to do well for many years after the subsidence of active rheumatic infection.

CASE 2. G. B. D., an 84-year-old man, entered the Phillips House on February 4, 1938, seriously ill, with a story of recent angina pectoris decubitus. The patient, always hale and hearty until old age, had been bothered for several years by a series of illnesses and accidents, beginning about 10 years previously with an automobile accident. Five years before admission to the hospital, he developed glaucoma and cataracts, and 3 years before, swelling of the legs from varicose veins began. There had been no dyspnea or chest pain until the summer of 1937, when he began to have discomfort in the upper chest on both sides of the sternum on effort; this was relieved by rest. The severity of the attacks gradually increased during the fall and winter. Three weeks prior to entry, the patient had been awakened at night by this same ache, which recurred through that night and at intervals on later nights. He had rested, but not in bed. Examination showed an elderly man, moderately ill, with normal blood pressure (145/80) and pulse rate (60). The heart was enlarged, with a total transverse diameter, by teleroentgenogram, of 17.5 cm. and a chest diameter of 31.0 cm. The electrocardiogram showed auricular premature beats with T_1 upright, T_2 low and T_3 inverted; there was moderate left-axis deviation, a PR interval of 0.20 second, and a normal Lead 4. A gastrointestinal x-ray series was normal.

Because this patient was already improving, without drugs, during the first few days in the hospital, no therapy except rest was prescribed. He improved steadily; in 2 weeks he was able to walk upstairs without symptoms, which he had not been able to do for weeks before, and not long thereafter he returned home free of symptoms *and without medicine*, much to the surprise of himself and of his family and friends.

In June, 1939, a close friend reported that the patient had been well after his return home in the winter of 1938 until the late winter of 1939, when he had a recurrence of his angina pectoris decubitus just like that of the previous year, which again cleared under the same therapy — rest without medicine, this time at home. In the summer of 1940, the patient was reported to be in good condition, even being able to attend evening meetings.

This case illustrates spontaneous recovery from severe coronary insufficiency — probably secondary to coronary thrombosis without infarction — twice at an advanced age. Rest, without drugs, doubtless helped in this recovery. The trouble with the eyes (glaucoma and cataracts) and with the legs (varicose veins) had restricted the patient's activities considerably, which may have prevented earlier symptoms of cardiac distress.

CASE 3. F. J., a stout, 61-year-old banker, in 1935, while sitting in a chair swallowing the first mouthful of a meal, fell unconscious without warning. The syncope lasted a few seconds. The patient had two similar spells in the next 2 days. He was then put to bed for 6 weeks, and reduced his weight from 246 to 226 pounds. An electrocardiogram showed right bundle-branch block but a normal PR interval. Two years later, in August, 1937, the patient fainted again, and thereafter was all right until October, when he began to have frequent attacks of faint spells and syncope. Despite much classic therapy, he continued to do very poorly, and was constantly threatened and actually affected by what was definitely proved to be Adams–Stokes attacks — the electrocardiogram during an attack showed a standstill of the ventricles, with a gradual return through partial grades of block to normal PR intervals. When he was at his worst, in March, 1938, the patient was having several attacks of unconsciousness daily. Physical examination showed no abnormalities, except for moderate obesity, a slight to moderate systolic murmur at the aortic valve area and at the cardiac apex, and occasionally an intermittent pulse, at a heart rate of 84. An electrocardiogram showed normal rhythm at a rate of 60 to 65, with right bundle-branch block and normal PR intervals. X-ray examination showed a full-sized heart, with a somewhat dense aorta.

The patient continued in a precarious state despite the use of atropine sulfate, nitroglycerin, ephedrine and injections of epinephrine hydrochloride intramuscularly — all of which did, however, undoubtedly help him tem-

porarily—during the next year. Finally, because of a critical sinoauricular heart rate of about 75, above which partial block and Adams–Stokes attacks occurred, the administration of digitalis was advised. Because of a previous unfavorable experience with digitalis, Urginin was given, and under its influence, in addition to that of adrenalin, he rapidly improved and during the next year and a half was in surprisingly good health, completely free from any attacks of Adams–Stokes syndrome.

Physical examination in November, 1940, showed moderate hypertension (200/90), a moderately enlarged heart with harsh aortic and moderate apical systolic murmurs and no evidence of congestive failure. On fluoroscopic examination, there was moderate enlargement of the heart, with clear lungs. The electrocardiogram showed complete auriculoventricular block, with an auricular rate of 85, a ventricular rate of 40 and bundle-branch block as before. The patient had reduced his weight to 216 pounds, and was quite active again, having resumed his regular work, though not intensely, at the age of 66 years.

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This patient showed a hopeful recovery from partial auriculoventricular block with Adams–Stokes syndrome. The transition to complete heart block was doubtless spontaneous and accounts more for his present state of good health than the drug therapy does. However, it is probable that the drugs helped him over a critical period. Although his duration of life is uncertain, it is hoped that he may continue in his present fair state of health for some years to come. We have encountered similar patients who have survived for ten years or more after the establishment of complete heart block.

The effect of sensible living, not only on the well-being but also on the longevity of the patient, is clearly demonstrated in the following group of cases. To prescribe drugs and to advise certain surgical procedures in many cases of heart disease are not only advisable but at times essential and useful. However, one should define to his patients, clearly and forcibly, the importance of personal readjustment to individual needs. Each case has to be worked out by itself. Often the patients themselves can determine their optimum mode of living, not only from the viewpoint of comfort, but also from that of earning a livelihood.

Case 4. A. E., an 82-year-old man, was seen in February, 1937, with cardiovascular limitation, both in the form of angina pectoris, helped at first by nitroglycerin, and in the form of congestive failure, which was greatly helped by digitalis. The patient suffered from progressive loss of vision, which began in 1910 and resulted in practically complete blindness in 1922, when glaucoma manifested itself; he continued to be active thereafter, however. In 1924, an electrocardiogram showed intraventricular block. The blood pressure was 170/110. The patient had dyspnea when he hurried, but his blindness prevented him from walking fast enough to cause much trouble with the circulation. In 1925 he developed angina pectoris, and the diagnosis of hypertensive heart disease was added to the previous ones. In 1926, definite evidence of congestive heart failure appeared, and the patient felt better when digitalis therapy was instituted. In 1927, he was given hypertonic salt solution intravenously, and this procedure was followed by an attack of coronary thrombosis. In the 10-year period following this attack, he suffered a good deal of cardiovascular limitation caused by angina pectoris, which was relieved by nitroglycerin, and by congestive failure, which was helped very much by digitalis.

The patient gradually learned how to improve his health by rest, breathing exercises and a careful diet. By this regime, his symptoms largely subsided in the course of years, so that he had almost no angina pectoris and little or no dyspnea, although it is true that he walked very slowly. He continued actively at work throughout the years, going to his office daily and resting almost completely in bed over week ends. On one occasion in the fall of 1937, he was referred to the Baker Memorial Hospital for rest on account of an increase in coronary insufficiency; this treatment resulted in considerable improvement. In February, 1940, he contracted bronchopneumonia and was admitted to the hospital. Although he seemed to weather the infection, he became very weak and died from a fresh coronary thrombosis 10 days after entry.

Autopsy. The heart weighed 500 gm. There was a mural thrombus in the left ventricle, with acute coronary thrombosis in the left descending branch, as well as a healed cardiac infarction, generalized arteriosclerosis and marked coronary sclerosis.

Here was an ingenious and wise man who taught his doctors how Nature, common sense, the affliction of blindness and personal experimenting aided longevity while he continued a useful life.

Case 5. M. P., a 43-year-old salesman, underwent a total thyroidectomy in January, 1934, to relieve severe angina pectoris of 6 months' duration. There was but little apparent improvement, and for the next 3 years the patient was unable to walk up a slight grade on account of chest pain. In view of a low basal metabolic rate (−32 per cent) and some evidence of myxedema, he took 1/10 gr. thyroid daily. For one year (1937–1938) he offered the information that his attacks were more marked after meals and dependent on the type of food ingested. After considerable experimenting with himself and with the lapse of time, he has been able finally to walk about 4 miles at a time without having an attack of angina pectoris, but he has learned to walk very slowly.

This patient's own comments on his well-being and freedom from attacks are interesting. Although the myxedema has had a quieting effect, he also has voluntarily slowed down, since his walking is methodical, never increasing the rate above 60 steps a minute. The evidence tends to show that a "slow pace in life" may allow a survival, at least in some cases.*

*Another case of coronary disease with severe angina pectoris treated successfully by total thyroidectomy in 1934 has interested us very much. A 67-year-old physician was treated by rest for five months without apparent benefit. After total thyroidectomy he was completely relieved of his angina pectoris, but he slowly developed myxedema. For this he took enough thyroid to hold his basal metabolic rate within normal limits. He felt well, and despite adequate thyroid in his system failed to suffer a recurrence of angina pectoris, except when he pushed himself hard. He is still carrying on general practice at the age of seventy-three years. It is evident that he had a natural recovery of coronary sufficiency, doubtless through the development of collateral circulation, with a tiding over—perhaps lifesaving—of a serious period of ill-health by the total thyroidectomy.

CASE 6. D. G., a 45-year-old jeweler was first seen medically in 1923. He had no complaint except that he had been rejected for life insurance on account of elevation of blood pressure, which was found to be 194/104. The patient was a dynamic person who enjoyed attending lodge meetings at least three or four times a week, and who took an active part in all the discussions. Although he was advised to "slow down," he refused to follow this advice, in view of the absence of symptoms.

The first attack of angina pectoris occurred in 1929, but in spite of this warning, the patient did not change his mode of living. Although he had no further attacks until 1934, the electrocardiographic tracings during the later years showed evidence of coronary-artery disease with myocardial involvement in the form of progressive changes in the T waves of Lead 1. The attack in 1934 lasted 2 hours and required morphine for relief, but was not convincing enough to cause a change in the patient's habits. However, a second attack of coronary occlusion accompanied by paroxysmal auricular fibrillation in July, 1935, completely changed his idea of how to live and be happy.

Five years have elapsed since the severe attack of coronary occlusion, and during this period the patient has resorted to the use of nitroglycerin on the average of about once in 2 weeks, when he finds himself driving his automobile in difficult traffic (this is not a safe thing for him to do). The physical findings during this 5-year period have not changed. The blood pressure has remained around 200/110. Recently the vital capacity was 2400 cc., and there was no evidence of failure. Treatment has consisted of 5 gr. of quinidine sulfate twice daily, 1½ gr. of aminophylline three times daily, and 10 minims of Lugol's solution once daily.

Since the attack in 1935, the patient has adopted a strictly followed routine. He arises at 7 a.m., gets his own breakfast and walks about ⅛ mile to the bus very slowly (he says, "I have plenty of time and never rush for an oncoming bus, for there will be another right along"). Up to January, 1940, he took rest periods in the afternoon for about 45 minutes or 1 hour; since then he has felt so well that he has given them up. He works until 5:30 or 6:00. His meals are light, with dinner at 6:30. Although he retires at 7:30, he reads much of the night or until he is drowsy. Once or twice a week he spends an evening at either a lodge meeting or visiting friends, but at all times returns home not later than 10:30. He never goes out on Sunday. Although his production, per working hour, has decreased, he has been able to meet his financial obligations and has maintained a living income.

Here is another example of what careful living, in addition to a natural tendency to longevity, can do.

CASE 7. M. B., a 48-year-old schoolteacher, consulted us on June 12, 1926, because of the recent onset of tachycardia and dyspnea. On physical examination, she showed moderate cardiac enlargement, with a well-marked, rolling, mid-diastolic murmur at the apex, accentuation of the first apical sound and of the pulmonic second sound and an apical third sound. The heart rate at the apex was 160, at the wrist 104. The electrocardiogram showed auricular fibrillation with slight right-axis deviation. A high degree of mitral stenosis was clearly evident.

Digitalization was begun and maintained thereafter. For 14 years, the patient has continued to teach, with occasional short pauses caused by acute illnesses or to recover from fatigue. Now, at the age of 62, she is ready to retire after a long, useful service. Despite increasing symptoms of pulmonary congestion and slight heart failure in the last 10 years, she has kept at work by the most careful regulation of her daily life. Her only activity has been to go to school, aside from brief vacation trips to the South in the winter or spring. She has rested during her long summer vacations. Often she has remained absolutely at rest after leaving school on Friday afternoon until she has gone again on Monday morning. She has survived several attacks of pulmonary infarction and of pneumonia and colds of lesser severity. For years she was able to drive her car but did very little walking. Only once or twice has she needed diuretic therapy, rest therapy and digitalis being adequate to prevent or control congestive failure.

When examined on June 15, 1940, the patient was in fairly good condition; she was convalescent from an illness of 7 weeks, probably precipitated by a small pulmonary embolism. She showed no evidence of heart failure. The heart rate was 56, and the rhythm irregular. The blood pressure was normal. The heart was large, with a loud systolic and a slight mid-diastolic murmur at the apex.

This case of rheumatic heart disease of more than average severity has permitted not only a considerable longevity but also a useful life of teaching through the carrying out of a sensible mode of life and the good fortune in being able to maintain a fair state of health and to avoid severe rheumatic infection. Familial longevity doubtless has entered into the picture.

DISCUSSION

Although this group of patients is small, it does represent a considerable number of cases of a similar character that physicians encounter during many years of experience. Often, however, the consultant may be unaware of the final outcome, nor does he always know what progress the patient has made subsequent to the consultation. It is important to recognize that the prognosis is as likely to be favorable as unfavorable in the majority of cases.

Three of the 7 cases reported showed serious heart trouble at the outset, with an unfavorable or even a grave prognosis; nevertheless, the patients experienced a natural or spontaneous recovery and did very well for a good many years. One of these patients was fortunate enough to have had an acute infection that saved him from a needless valvotomy, with a possibly fatal outcome.

In the second group were 4 patients, 3 of whom were able to continue to earn a livelihood for many years in spite of serious heart disease. This was accomplished undoubtedly by the careful program that each one mapped out for himself. The fourth patient, although he was unable to return to his original occupation, could without question employ himself in a less vigorous trade than that of a traveling salesman.

Summary

We have presented 7 cases of severe heart disease in which the patients, who at the onset of their illness appeared to have unfavorable prognoses, but through natural recovery or sensible living, were able to lead long and useful lives. This group emphasizes the statement that in acute heart disease "functional recovery may be so complete that the ultimate prognosis is good for many years after." It is not to be inferred, however, that medical attention can be dispensed with. Furthermore, every person should seek medical counsel at the onset of symptoms, whether or not they are of cardiac origin.

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THE TREATMENT OF HAND INJURIES*

Sumner L. Koch, M.D.†

CHICAGO

THE basic surgical principles involved in the treatment of compound injuries are nowhere better exemplified than in the painstaking and purposeful treatment of injuries of the hand.

Treatment that ensures arrest of bleeding and protects the open wound from further contamination constitutes ideal first-aid care. Most cases require only the immediate application of a sterile dressing, held in place with the moderate pressure of a snug bandage and in severe cases reinforced with a light splint. The more promptly such a dressing can be applied, the less is the danger of contamination of the open wound with virulent organisms. With increasing experience, surgeons have come to realize that the most important potential sources of such organisms are the uncovered mouths and noses of excited and voluble bystanders, or of any persons who may be attempting to apply some form of emergency treatment without first masking their faces. In most cases of compound injury, uncovered hands, unmasked faces and instruments hastily sterilized by rinsing them in some antiseptic solution carry a greater menace for the patient than the instrument that caused the wound or the bacteria that may have been present on the patient's skin at the time of injury.

A second principle, sometimes forgotten until too late, is that diagnosis should always precede treatment. Every surgeon of experience knows how little information can be obtained by inspection or probing of an open wound. The hasty operator may forget that accurate and important information can be quickly obtained by examination of the function of the part below the site of injury. Division of the ulnar or median nerve, for example, or of the flexor tendons of thumb and fingers can be recognized with certainty by simple tests that every first-year medical student knows full well and that too many residents and house officers have forgotten.

Once the extent of the injury has been determined, the next question is to decide what should be done immediately. Should cleansing of the wound be followed by operative repair of the injured structures, or should such repair be deferred until the question of wound infection can be answered with certainty? The time that has elapsed, the place where the injury was sustained, the first-aid treatment, the character of the injury itself and the facilities available for repair all come into consideration. Rarely would one attempt operative repair of an extensive wound if more than two hours had elapsed. Wounds sustained out of doors, in an automobile accident or in the street are likelier to be seriously contaminated than those sustained in a clean factory, a workroom or a kitchen. Careless or unthinking first-aid treatment may permit secondary wound contamination that leads to serious infection. Crushing injuries and deep and extensive lacerations are more difficult to transform into clean surgical wounds than clean-cut incised wounds caused by glass or sharp metal. The attempt to repair a serious injury in an emergency or a ward dressing room without adequate assistance is almost certain to result in disaster. Difficult surgical procedures demand every advantage that a well-equipped operating room and expert assistants can provide.

Whether or not operative repair of injured tissues is to be undertaken immediately, every wound should be carefully and thoroughly cleansed at the earliest possible moment to convert the contaminated wound into a clean wound. To secure

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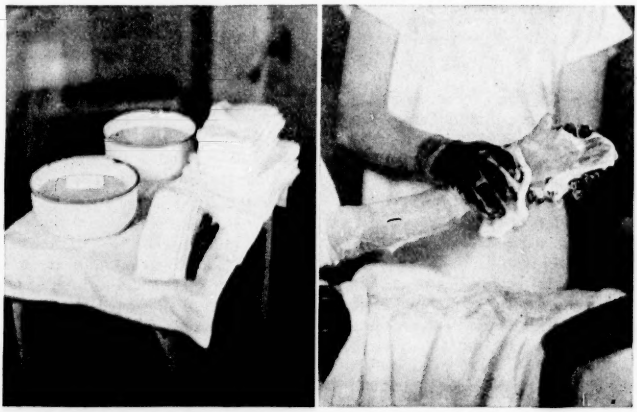
this result, I know of no more effective method than patient and thorough cleansing with ordinary white soap and sterile water, applied with soft sterile cotton and hands covered with sterile gloves, under eyes that are alert and mouth and nose that are completely masked (Fig. 1). Soap-and-water cleansing of at least ten minutes' duration is followed by thorough and prolonged irrigation of the open wound and all its recesses with warm physiologic saline solution. Charity may cover a multitude of sins, but the hope that sulfanilamide crystals can cover a multitude of virulent organisms, which may have come from the open mouth or uncovered nose of well-intentioned persons giving first aid, and completely neutralize their harmful effect is certain to result in disappointment. The value of chemotherapy should not be minimized, but the relative importance of the ounce of prevention and the pound of cure is just as great in wounds of the hand as in any potential disaster.

Time and space do not permit the discussion of many details that are essential in actual surgical treatment, or of the basis on which conclusions that may seem too dogmatic are founded. Nevertheless these conclusions are based on a considerable experience and a constant search with an open mind for the best method of meeting the problems in question. That there must be one most effective technic in treating open wounds in spite of the widely divergent methods and ideas in vogue whenever compound injuries are treated, no one would deny. Tomorrow may bring improved methods, but the immediate problem is to accomplish the best possible results with the facilities available today.

Briefly, then, the treatment of patients with hand injuries should include the following: general anesthesia in cases of severe injury; a bloodless field secured with the aid of a blood-pressure cuff inflated to 250 mm. of mercury; incisions that permit adequate exposure of injured tissues, retracted muscles and tendons and that do not cut across flexion creases (Fig. 2); gentle handling of tissues, no matter how much greater the expenditure of time; accurate hemostasis; the use of silk ligatures and sutures of the finest caliber that afford the required tensile strength; reduction of fractured bones without interposition of foreign material; repair of divided tendons by a method that leaves the tendon ends as free as possible from the irritating action of foreign material; suture of divided nerves with the finest possible silk suture that includes in its



A B

FIGURE 1. *Preparation of the Field of Operation.*

A—a small table, with two basins of sterile water, a pile of soft cotton, a cake of plain white soap, a towel and dry sterile gloves, is ready beside the operating table. B—the cleansing of the field of operation is gentle, but thorough and painstaking, and is continued for at least ten minutes (Koch[1]; reproduced by permission of the publisher).

bite only the epineurium (Fig. 3); and, finally, complete and accurate wound closure without drainage. These details of treatment are carried

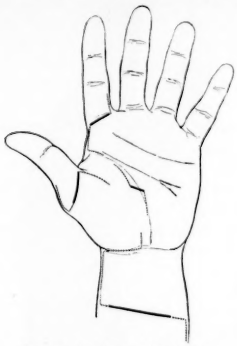


FIGURE 2. *Lines of Incision.*

Incisions for securing adequate exposure should be made to lie parallel with flexion creases and not across them. Median incisions of the fingers and forearm and cruciate incisions should always be avoided (Koch[1]; reproduced by permission of the publisher).

out not in blind adherence to a routine inherited from the past, but as the result of a serious effort to learn by experiment, by observation of one's own results and those of others and by thoughtful consideration of what methods afford the best possibility of securing the desired result of healing with a minimum loss of time and function.

Perhaps no one of these details is likelier to arouse controversy than the suggestion to close the wound accurately and without drainage; yet it is just as vital to secure healing by primary union for the patient who has sustained a division of nerves and tendons as it is for the patient whose hernia has been repaired or whose stomach has been anastomosed to the small bowel. It is even more important, since any wound infection, no matter how trivial, impairs the usefulness of structures, such as tendons and joints, the function of which depends on complete freedom of movement. I should even say that if the wound cannot be closed accurately and without tension because of loss or destruction of covering tissue, it

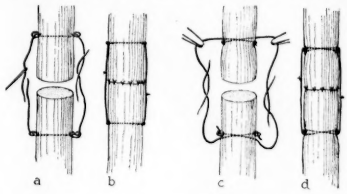


FIGURE 3. *Technic of Tendon Suture (Mason).*

Both methods (a and b, and c and d) are consistent with the important criterion that the free ends of the two segments be as free as possible from foreign material. Experimental studies indicate that in the second method (c and d) there is less tendency for the tension suture to cut through the tendon and permit partial separation of the sutured ends (Koch[1]; reproduced by permission of the publisher).

FIGURE 4. *Crushing Injury of the Hand Treated by Immediate Soap-and-Water Cleansing, Wound Repair and Immediate Covering of the Extensive Raw Surfaces with Grafts of Intermediate Thickness (patient of Dr. Michael Mason).*

Appearance of the hand before operation.

is the obligation of the surgeon to provide covering tissue by the use of a flap or graft, so that the desired result of immediate wound closure can be attained (Figs. 4, 5 and 6). If the use of a flap or graft is necessary, one rarely undertakes repair of divided nerves or tendons until wound healing is complete. The latter procedures do not demand immediate attention, but to miss the most favorable opportunity to secure closure of a wound with the aid of a graft or a flap where there has been extensive loss of covering tissue and to await the formation of granulation tissue and the crippling fibrosis invariably associated with it before attempting to secure wound closure are misfor-

FIGURE 5. *Crushing Injury of the Hand Treated by Immediate Soap-and-Water Cleansing, Wound Repair and Immediate Covering of the Extensive Raw Surfaces with Grafts of Intermediate Thickness (patient of Dr. Michael Mason).*
Appearance of hand at the primary dressing.

FIGURE 6. *Crushing Injury of the Hand Treated by Immediate Soap-and-Water Cleansing, Wound Repair and Immediate Covering of the Extensive Raw Surfaces with Grafts of Intermediate Thickness (patient of Dr. Michael Mason).*
Hand immobilized with aluminum splint and enveloped in a large gauze dressing, bandaged under moderate pressure, and left undisturbed for fourteen days after operation.

tunes for both the surgeon and the patient.

When the wound has been closed, whether by suture or graft, a large pressure dressing consisting of fluffed gauze, cotton waste or sea sponges is applied over the entire wound area, and the part immobilized with the aid of a light aluminum splint. Inflamed and injured tissues need rest, and the rest secured by the compression bandage and splint is uninterrupted, if possible, until the time when the sutures should be removed. If the treatment has been painstaking and well performed, one may wisely leave the dressing and wound untouched. The patient is watched carefully for evidence of impaired circulation and wound infection; the dressing is left undisturbed.

Finally, one cannot refrain from emphasizing the oft-repeated precept: The outcome of any case depends primarily on the surgeon who first sees the patient. If his treatment is skillful and well considered, rarely will anything more need to be done at a later date, and if further treatment is necessary the conditions will usually be favorable. If the treatment rendered immediately after the injury is haphazard and thoughtless, it too often happens that no surgical care, no matter how skillful or painstaking, can compensate for the initial neglect of the basic principles of treatment of compound injuries.

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MEDICAL PROGRESS

KIDNEY DISEASE

Reginald Fitz, M.D.*

BOSTON

SINCE my last review,[1] the varying problems of Bright's disease have continued to be attacked in many ways: by clinicians who have attempted to observe patients at the bedside more accurately than heretofore; by laboratory workers who have devised ingenious experiments to produce, prevent or cure experimental nephritis; and even by surgeons who have tackled the lesions of nephritis by surgery rather than by more orthodox medical methods. Hence any reader in this general field of medical literature soon has his appetite surfeited. To avoid mental indigestion, he is driven, perforce, to include in his diet only those literary delicacies that seem most digestible for his particular type of mind. This year, therefore, I have made no attempt to summarize all the communications of the last twelve months but instead have selected for discussion a group of papers that I have found both readable and stimulating.

One of the ideas that nowadays attract almost every clinician is the thought of being able to use plasma in some form for purposes of blood transfusion. The possibility of utilizing concentrated plasma-protein solutions in the management of renal edema associated with reversed albumin-globulin ratios and lowered blood-protein concentrations springs naturally to mind. Thus the observations of Aldrich and Boyle[2] are important as being among the earliest on record in this particular effort. These workers used concentrated solutions of serum protein as a diuretic in a group of patients with nephrosis. They dehydrated pooled serum to make its normal protein concentration four times normal, and injected the resultant solution by vein in doses that varied from 25 to 65 cc. In most of their cases, diuresis followed such treatment and continued until the edema was eliminated. The possibilities of developing this method of therapy in certain types of renal edema are promising: concentrated plasma, in fact, may prove to be an excellent diuretic not only in renal edema but also in other conditions in which water retention depends on disturbed physicochemical relations in the blood and tissues.

A few years ago a charming doctor from Dublin named O'Shaughnessy visited this country. He spoke in New England at several of our local medical meetings — a fiery, enthusiastic young Irishman, sincerely interested in the possible value of omentopexy for the treatment of angina pectoris. His idea was to bring a piece of omentum through the diaphragm, attach it to the heart apex and thereby stimulate the formation of a new blood supply to the myocardium. His work did not stir up great excitement in Boston, but everyone who heard O'Shaughnessy speak was impressed by his sincerity and uprightness. The announcement of

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his death on active service with the British Expeditionary Force in Flanders made all his American friends feel badly, for no one could meet him without hoping to see him again. But at least his ideas carry on. MacNider and Donnelly,[3] modifying his method, have attempted to establish in animals an adventitious circulation in the kidney by omentorenopexy, and Weeks, Steiner, Mansfield and Victor[4] appear to have successfully treated experimental renal hypertension by the operation of splenorenopexy. By the last procedure, it seems, collateral circulation between the splenic sinusoids and the capillaries about the renal tubules was provided, with an improved circulation through the kidneys and a resultant fall in blood pressure. When the fused spleen and kidney was later removed, the blood pressure soon returned to its previous level of hypertension. Perhaps eventually we shall hear more of the clinical usefulness of O'Shaughnessy's ideas and of their application in directions other than he supposed.

Goldblatt's work continues to inspire clinicians to hunt carefully for patients with hypertension whose abnormal blood pressure level is caused by unilateral renal conditions. A variety of new cases have been reported: perhaps the most peculiar one is that described by Koons and Ruch,[5] which concerns a seven-year-old child with a Wilms's tumor complicated by hypertension. The tumor did not appear to be the source of the hypertension since, when an exploratory operation was performed, extensive metastases were seen and felt, and these presumably would have perpetuated hypertension after nephrectomy if the tumor had manufactured any pressor substance. Yet actually the hypertension disappeared when the kidney and the primary tumor were taken out. Koons and Ruch explain this happening on the assumption that, since the tumor practically surrounded the kidney, it caused renal ischemia and acted in much the same way as the cellophane membrane that Page and his associates[6] placed around the kidney in animals to produce experimental hypertension. If this explanation is correct, this is the first clinical confirmation on record that Nature has a means of duplicating in man so bizarre a renal lesion as the one made synthetically by Page in the laboratory.

Many typical cases of unilateral atrophic pyelonephritis cured temporarily, at least, of hypertension by nephrectomy are now on record, so that an increasingly large group of such patients are being followed for lengthening periods. A patient of mine,[7] whose case was reported in 1940, continues to have normal blood pressure; to the list have been added during the past year a Canadian case reported by Patch, Rhea and Codnere[8] and 5 new cases reported by Barker and Walters,[9] of the Mayo Clinic. Everyone seeing these cases maintains a proper attitude of conservatism over end results. Palmer, Chute, Crone and Castleman[10] express the generally accepted viewpoint: for success in this operative approach to hypertension, only those cases must be selected for surgery in which there is no recurring pyelitis or other abnormality on the apparently normal side.

Neurosurgeons have become a little less vociferous than they have been in previous years. To be sure, Peet, Woods and Braden,[11] staunch proponents of such complicated operations as bilateral supradiaphragmatic splanchnectomy with lower dorsal sympathetic ganglionectomy, have reported results in 350 cases. They claim that in slightly over half the cases in their series there was significant reduction in blood pressure and that after operation, clinically speaking, their patients' disability was improved in the vast majority of cases. This is a good record, which speaks for itself. On the other hand, Davis and Barker,[12] of the thiocyanate school, claim that bilateral splanchnic section fails to give relief to patients who cannot obtain equal benefit from the proper use of this drug. They still maintain that many patients with hypertension respond favorably to thiocyanate and that the drug is not unduly dangerous, and apparently, they continue to use it with satisfaction. The perfect treatment of hypertension is still undiscovered.

When the American College of Physicians met in Boston this spring, no single paper was read that provoked more discussion in the corridors, bedrooms and dining rooms of the Hotel Statler than that of Page and his associates[6] on the use of kidney extract in the treatment of experimental and clinical hypertension. Beforehand, everyone wondered how Page would describe his most recent work, and afterward, how convincing were the results reported.

Page has elaborated a theory that is interesting. He believes that the diseased kidney may produce a substance which raises blood pressure and that this, in turn, can be neutralized and rendered inert by an antisubstance developed in the normal kidney. With this thought in mind, he at first carried out experiments on animals in which extracts from normal kidneys were used in an attempt to counteract experimentally produced hypertension. Elevated blood pressure was induced by several different means and appeared controllable by properly graduated doses of normal renal extracts. These results were sufficiently convincing to Page to make him try the next

hurdle. At the meeting of the College of Physicians, he reported on 19 human cases of hypertension treated in similar fashion. He drew no conclusions that were at all unguarded. However, he produced evidence to suggest that the parenteral injection of renal extract in his cases appeared, on the whole, to have been of some value in treatment. In certain cases, for example, there was marked improvement in the condition of the eyegrounds, in the cardiac symptoms and in the blood-pressure levels. On the other hand, severe reactions occasionally developed, owing, he believes, to the protein content of the extracts employed. He concluded his remarks, wisely and sanely, by stating that the use of kidney extracts in the treatment of certain cases of hypertension probably has some merit and at least deserves further study. Knowledge concerning the true usefulness of the technic or even of the idea behind it is too fragmentary to bear much discussion. Yet no one who heard Page speak could fail to believe that his work deserves watching.

When one leaves the laboratory and turns more closely to the bedside for the basis of his reading, he encounters several worthwhile papers. Lupus erythematosus is much in the public eye at the moment. Hence the paper by Stickney and Keith[13] is interesting. They studied the kidneys in 15 fatal cases and concluded that there is no distinct renal lesion peculiar to the disorder. In 8 of their cases, the kidneys appeared normal; in 2, glomerulonephritis was found; and in 5, there was no more specific form of nephritis than a certain amount of proliferation of the glomerular endothelium, together with irregularity and thickening of its basement membrane. Thus lupus erythematosus cannot be regarded as having any typical form of kidney lesion attached to its pleomorphic pathology.

The relation of tonsillar infection to nephritis has been discussed perennially for a long time. Illingworth,[14] in England, is the latest clinician to claim that tonsillectomy neither cures nephritis nor prevents its progress; in fact he even goes so far as to suspect that tonsillectomy may predispose to nephritis or may actually precipitate an attack. The paper was printed a year and a half ago, and his ideas seem not to have been repudiated or generally supported by his British colleagues.

Acute nephritis and the acute exacerbations that so often occur in the course of chronic nephritis have also received due consideration. Hayman and Martin,[15] in an excellent paper, have analyzed the histories of 22 cases of acute nephritis. The most interesting point in their paper is that about two thirds of their patients recovered completely. Acute nephritis, too, as they point out, is, on the whole, a disease of young people and, contrary to Illingworth's views, is likely to be precipitated by acute upper respiratory-tract infections involving the throat, sinuses and ears.

That acute glomerulonephritis may follow so simple a skin infection as impetigo is emphasized in a paper by Futcher.[16] He picked out of the Johns Hopkins Hospital Clinic 11 cases in which glomerulonephritis followed impetigo or some other superficial skin infection, although, to be sure, beta-hemolytic streptococci were not infrequently found in these cases on throat as well as on skin culture. That skin infections may be followed by so serious a complication as glomerulonephritis in certain cases is worth remembering.

Seegal, Lyttle, Loeb, Jost and Davis[17] have reemphasized the relapsing character of chronic nephritis. They say that the life history of a patient with chronic nephritis is often punctuated by repeated attacks of acute nephritis superimposed on the basic underlying chronic lesion. Such attacks are usually accompanied by transient decreases in renal function or by sudden marked hematuria. One of their patients with chronic nephritis had, they believe, several attacks of acute nephritis in the course of a few years. This paper is an interesting attempt to establish the complete picture of chronic nephritis through utilization of the method of careful follow-up studies on a small group of patients observed over a long period. To my mind their ideas carry considerable weight. It is highly probable that repeated acute insults, each with or without complete healing, do much to account for the characteristically slow progress of many cases of chronic Bright's disease.

Now that the ordinary clinician is becoming educated to recognize gout more readily than he used to, gouty nephritis is again receiving attention. Talbott and his co-workers[18] at the Massachusetts General Hospital have written a clever paper to remind us of what our English forbears knew so well: that most subjects with gout show some evidence of renal damage. Certainly they have performed some painstaking and superb studies on earmarking the characteristic renal lesion of gout — clinical studies that would have amazed old worthies like William Cadogan or Benjamin Franklin, who thought they understood the gouty diathesis. The urates seemingly can be filtered through the gouty kidney but are reabsorbed with difficulty. Drugs like cinchophen and Salyrgan depress further urate resorption but stimulate urate clearance; colchicine, strangely

enough, has no particular effect one way or the other, so that at present why it appears to behave so beneficially in gout is uncertain.

The sulfonamides are so popular that naturally they have been used abundantly in the treatment of various urinary infections. Cook[19] has written a helpful paper describing which ones of the new members of the family are of particular help in combating genitourinary infections caused by a variety of different organisms. It is obvious that the causative agent must be established and that each case is best treated individually. In Cook's experience, sulfanilamide, dimethyldisulfanilamide and sulfanilylsulfanilamide were most effective against gram-negative bacilli, beta-hemolytic streptococci and the proteus bacillus, whereas sulfapyridine seemed more useful to combat staphylococcal infections. Sulfathiazole proved useful against gram-negative bacilli and staphylococci but was not very efficacious against beta-hemolytic streptococci. Finally, when the renal function was poor, azosulfamide seemed to be the drug of choice since it appeared to have little or no injurious effects on the kidney. In this connection it is worth remembering that all members of this family, even when properly used, may bother the renal system. The sulfonamides tend to precipitate in the collecting tubules or even in the ureters and bladder unless due care is taken, and thus they act as renal irritants. I like Arnett's[20] rule in using these drugs—a minimal intake of 3000 cc. of fluid each day, with an output of 1500 cc. or more, and careful watching of the urine for the presence of hematuria. No patient enjoys having to put up with urologic manipulation to rid himself of the chemical calculi that may form when he uses these medicines.

* * *

On the whole, the past year has been one of activity in the advancement of knowledge regarding Bright's disease and its treatment. The trend of the times is to pay increasing attention to renal physiology, so that a great deal of work is now going on with pressor substances and their relation to hypertension. On the other hand, clinicians still continue to see cases of chronic nephritis frequently and to hope for the development of more secure methods for the cure and prevention of this disease than now exist.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27291

PRESENTATION OF CASE

A seventy-two-year-old man entered the hospital for study.

The patient was seen for the first time by a staff member of this hospital nine years before admission, when diagnoses were made of irritable heart, — possibly on the basis of coronary disease, — auricular fibrillation and myxedema. Shortly afterward, he was confined to bed for twelve weeks, suffering from what was either pulmonary infarction or an incidental pulmonary infection. Seven years before admission, he had an attack of dyspnea, which lasted fifteen minutes and was not accompanied by pain, palpitation or cough. He remained in bed for three days, after which he returned to his usual activities. Apart from occasional shortness of breath, he remained well for the next seven years, and during the summer preceding his admission he played golf without untoward effect. Four months before entry, the patient suffered a violent nosebleed, and a month later noticed the onset of gradually increasing dyspnea, even while walking on level ground. A severe attack of dyspnea developed during a walk ten days before admission; it lasted ten minutes and, again, was not accompanied by other symptoms.

The patient had been constipated for some time and had noticed an urgency and frequency of urination, with the passage of small amounts, and a nocturia of three times a night. Several days before entry, a dry cough had developed.

When thirty-two years old, the patient had suffered from mumps complicated by severe bilateral orchitis. Shortly after this episode, he noted marked loss in sexual power, which proceeded to complete impotency. His skin became fine in texture, his beard regressed, the pubic and axillary hair fell out, and his phallus decreased in size. There was no marked loss of strength or of weight. He did not experience hot flashes at any time. It was thought by some that the pitch of his voice became higher.

He had four children, who were living and well.

On physical examination, the patient's facies was definitely eunuchoid in appearance; it also had somewhat the appearance of that seen in myxedema. The skin as a whole was of fine texture, the speech was slow, and the hair of the head was normal but that of the extremities, axillas, pubic region and perianal region was absent; the testes were small; no prostate tissue could be felt.

The neck veins were moderately distended, with visible pulsations. The left border of the heart was 3 cm. outside the mid-clavicular line, — the heart was appreciably larger than it had been nine years previously, — and a soft systolic murmur was heard at the apex and to the left of the sternum. The aortic second sound was greater than the pulmonic. The rhythm was irregular, the blood pressure 160 systolic, 90 diastolic. A few rales were present at the right lung base. The abdomen was normal. There was a minimal pitting edema of the shins.

The temperature was 98°F., the pulse 88, and the respirations 20.

The urine showed a + test for albumin. The blood showed a red-cell count of 4,520,000 with a hemoglobin of 13.4 gm. (photoelectric-cell technic), and a white-cell count of 8400. The blood sugar was 82 mg. and the cholesterol 101 mg. per 100 cc. The 17-ketosteroid excretion in the urine was very low, but not absent, on two occasions; the actual figures were 1.4 and 1.2 mg. per twenty-four hours. There was no excess of follicle-stimulating hormone in the urine; the basal metabolic rate was -15 per cent.

An x-ray film of the chest showed the diaphragm to be high on the right side and its respiratory movements limited. There was mottled dullness along the right border of the heart, and in the angle between the heart and diaphragm; the costophrenic sinus on the right side was shallow. The heart shadow was considerably enlarged in its transverse diameter, and there was a definite bulge of the right auricle. The supracardiac shadow was not increased, the appearance of the aorta was not remarkable, and there was no visible calcification or mediastinal masses. X-ray examination of the skull showed the sella turcica to be rather large and bridged, but no abnormalities of shape or erosion of the clinoid processes could be seen. The pineal gland was visible and in the usual position. The bones of the skull were unusually heavy. There was no evidence of increased intracranial pressure, and the usual blood-vessel markings were present.

The electrocardiogram showed an unusual ectopic auricular rhythm, regular at 160 or more; this was considered to represent an ectopic auricular tachycardia. For the most part, there was a

2:1 ventricular response, with an occasional ectopic ventricular contraction. The ventricular rate was 85; T_1 was low, T_2 slightly inverted, T_3 inverted. Q_3, which varied from 2 to 3 mm. in length, was inverted, R_4 upright, and T_4 low.

The patient was given digitalis; the heart slowed but remained irregular, with a varying degree of auriculoventricular heart block. The edema had cleared on the fourth hospital day. Ten days after admission, the patient was started on testosterone propionate, 12.5 mg. intramuscularly three times weekly. This therapy was continued until three days before death. At two weeks, a chest plate showed that the process in the lungs had cleared; the diaphragm was in the usual position, and the lung fields were clear; the heart shadow was roughly triangular and was increased somewhat in size, the increase being largely to the right.

The patient was discharged under the care of his physician.

He made a follow-up visit two months after discharge. At that time, everything seemed to be going very well. He felt considerably stronger; he had noted a return of sexual power.

At 4:00 in the morning after the follow-up visit, the patient's wife was awakened by nightmarish noises from her husband. She failed to arouse him and finally called his physician, who arrived at 5:30 a.m. The patient was in moderately profound coma, with Cheyne–Stokes breathing and pinpoint pupils, but without obvious paralysis. The blood pressure was 200 systolic, 90 diastolic, and the pulse was 60. Three hours later, his condition was essentially unchanged, so that aminophyllin was administered, with the result that breathing improved and the patient roused to the point of movement and answered a few simple questions, although his words were scarcely intelligible. That evening, another administration of aminophyllin wrought no change; there was urinary incontinence, respiration had improved, the pulse was irregular at 80, the blood pressure was 185 systolic, 90 diastolic, and the pupils were constricted.

The following day, the patient was still semiconscious and incontinent, with Cheyne–Stokes breathing, a blood pressure of 150 systolic, 75 diastolic, and a pulse of 60. That evening, coma deepened, the temperature was 102°F., the pulse was faster, and the patient seemed choked up.

The next day, the temperature was 104°F., the respirations 50, and the pulse 100. Coma was even more profound, and diminished breath sounds and rales were heard throughout the right chest. The patient died that day.

Differential Diagnosis

Dr. Edwin B. Astwood:* We have, in brief, a seventy-two-year-old man who, at the age of thirty-two, had a severe orchitis as a complication of mumps, followed by complete sexual impotency and certain other endocrine changes, but who remained well until nine years before entry, when he began to have symptoms referable to his heart. He finally entered the hospital for study, presumably because of increasing dyspnea and signs at that time of moderate cardiac decompensation. This cleared on digitalis and rest, and he was discharged well. He died suddenly, or at least died very shortly after a sudden attack, apparently of something entirely different. I do not believe that one single disease can explain everything. We might briefly discuss the three aspects that are most outstanding.

In the first place, the cardiac condition was longstanding, nine or ten years, and was accompanied by cardiac enlargement and finally by symptoms of failure, with the presenting symptom of dyspnea. I do not consider the mention of myxedema sufficient to account for the enlargement of the heart, or definite enough to account for the cardiac condition. The presence of dilatation of the right auricle, taken together with the ectopic auricular tachycardia, might indicate that there was myocardial damage in the right auricle. Auriculoventricular block also suggests myocardial damage, and in a man of seventy-two the most probable diagnosis is a coronary arteriosclerosis, with possibly myocardial infarction. I do not believe that the electrocardiograms are contradictory to such a diagnosis. When the patient entered the hospital, the physical signs were in keeping with an increased venous pressure, with pulmonary congestion and perhaps with pleural fluid and enlargement of the liver. This condition cleared on rest and digitalis, and probably represented a mild cardiac decompensation.

The cause of death, however, seems to have been a second type of condition. If the first assumption — that the patient had coronary arteriosclerosis — is correct, it is reasonable to assume that he had arteriosclerosis elsewhere, although no mention is made of it. He did have hypertension, and sudden coma coming on in a hypertensive man of seventy-two is suggestive of a cerebrovascular accident. The absence of localizing signs and paralysis indicates not a corticostriatal lesion, but perhaps a thalamic or mid-line lesion. A lesion in the pons might be more probable because of the pinpoint pupils and final hyper-

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pyrexia. Whether the condition was thrombosis, embolus or hemorrhage rests on more details of the actual events leading up to the accident. This patient with complete impotency for forty years had a sudden restoration of sexual activity, and encountered an eventually fatal coma; this, I think, strongly suggests cerebral hemorrhage, possibly in the pons.

The third point to be considered is the nature of the endocrine disturbance. We are told in the history that the patient had mumps complicated by bilateral orchitis, and that this was the cause of loss of sexual power and loss of hair. There are several points that might be against such a concept. In the first place, it is very rare indeed for this type of orchitis to produce complete destruction of the testicular tissue. Experimentally, 2 per cent of viable testis tissue is sufficient to allow fairly normal endocrine function of the testis. Furthermore, complete azoospermia following such an orchitis is extremely rare, and still rarer would be complete destruction of the interstitial elements. Another thing against the possibility that the testicular atrophy was due to this cause is that the patient had complete impotency, which is scarcely credible even on the basis of complete castration. The loss of all body hair is something one does not usually associate with loss of testicular function. These three points are better explained by pituitary insufficiency, because all are symptoms of Simmonds's disease. This is not complete hypophyseal deficiency, however, because the patient maintained his weight and noted no loss in strength. There are a few other things that would support the concept of pituitary damage. The low metabolic rate, -15 per cent, and the myxedematous appearance, without real myxedema — the skin was soft and smooth, and the cholesterol value was 101 mg. per 100 cc. — one would associate not with myxedema but with hypothyroidism of hypophyseal origin. The low 17-ketosteroids, as Dr. Albright has shown, could mean that he was an old and debilitated man suffering from some nonspecific illness associated with an absence or a great reduction of adrenocortical function and an absence of testicular function. All this endocrine disturbance may indicate a man of seventy-two who had no testis function, and the other endocrine changes may have been the result of old age. However, I should think that the major endocrine disturbance was hypopituitarism, and although I have never heard of such a sequela, the change apparently occurred at the time when he had mumps; it is possible that it might have been a complication of mumps, just like other glandular complications, or was perhaps related to meningitis.

In summary, I should consider the cardiac condition as coronary sclerosis, with infarction; the terminal event, cerebral hemorrhage, perhaps pontine; and the endocrine disturbance, hypopituitarism.

Dr. J. H. Means: I saw this patient in consultation, and the question that was put up to me was as follows: "This patient has had myxedema for some time and appears to have a combination of myxedematous heart and coronary disease, with arrhythmia. Will you please see him and advise concerning treatment for hypothyroidism?" Because of his heart disease, they thought that they ought to go cautiously with thyroid. I saw him and did not think he had myxedema. I was impressed more than anything else with the factor of eunuchoidism. The man was emphatic in saying that this dated from the mumps. I was not certain whether anyone could get complete destruction of the testes from such an orchitis. I raised that, however, as one possible explanation, but also, like Dr. Astwood, suggested that it could be, and more probably was, on the basis of hypopituitarism.

One point about the diagnosis of myxedema: if this patient had had ordinary myxedema nine years before entry, and had not had treatment with thyroid in the interim, it would be impossible to have a picture of this sort. He would have been in an advanced stage of classic myxedema by the time of admission. The longest we have known any patient with myxedema to go is fifteen years — that is the utmost life expectancy. But after nine years with no appreciable change, we can say that he certainly did not have any ordinary myxedema.

One other point that should be brought up is whether the therapy in any way contributed to his demise. I suggested that it might benefit him, but I now believe that this may have been bad advice.

Dr. Fuller Albright: I went through the same reasoning as Dr. Astwood did and came to the same conclusion. I should emphasize one other bit of evidence, which Dr. Astwood did not make use of: there was no excess of follicle-stimulating hormone in the urine. If the patient had had complete destruction of his gonads by mumps and if there was no pituitary disease, he should have had a positive follicle-stimulating-hormone test. The fact that it was negative is strong evidence that the pituitary gland was the origin of the disturbance.

The question has been raised about the rationale of administering testosterone in this case. It is our belief that testosterone has a much wider effect on the patient than merely to stimulate sec-

ondary sex characteristics. I refer especially to its effect on nitrogen metabolism, which consists in a marked sparing of protein. Unless there are some definite contraindications, I am inclined to believe that patients with lack of testosterone production should receive this hormone. Furthermore, we have found in patients with panhypopituitarism — that is, underfunction of all parts of the anterior portion of the pituitary gland — that testosterone has a markedly beneficial effect.

The question has also been raised whether testosterone therapy in any way led to the patient's death. One must probably proceed with caution in administering testosterone to elderly patients for fear that the increased libido might lead to excesses, which might be bad for their cardiovascular system. In this particular case, no such increase in libido resulted, and I think we can dismiss this possibility. Furthermore, the symptoms at the time of death do not convince me that the primary cause of death was coronary thrombosis.

One other possibility occurs to me. Patients with panhypopituitarism have a marked tendency to hypoglycemia. Testosterone therapy, by inhibiting the conversion of proteins into sugars, might easily increase this tendency. I should at least raise the question whether the beginning of the acute symptoms that led to his death was not hypoglycemia. The semicomatose condition in which he remained for two days is most suggestive of this condition.

Clinical Diagnoses

Coronary heart disease.
Auricular tachycardia and fibrillation.
Hypogonadism.

Dr. Astwood's Diagnoses

Coronary sclerosis, with infarction.
Cerebral hemorrhage, probably pontine.
Hypopituitarism.

Anatomical Diagnoses

Coronary sclerosis, with fresh thrombosis, left descending branch.
Adenoma of the pituitary, neutrophilic type.
Hypopituitarism.
Atrophy of the thyroid gland.
Atrophy of the testes.
Chronic fibrous orchitis.
Hypertrophy of the heart.
Eunuchoidism.

Pathological Discussion

Dr. Tracy B. Mallory: The post-mortem examination showed obvious heart disease. The heart weighed 560 gm. The patient had severe coronary sclerosis, and there was fresh thrombosis of the left descending branch, which had not as yet led to any gross infarction. On anatomic grounds, this was the only possible cause of death discovered, because in the brain there was no hemorrhage or evidence of thrombosis or cerebral softening. Of course, I cannot rule out hypoglycemia, and Dr. Albright makes out a strong case for it. The testes were a little small, evidently fibrosed. On microscopic examination, the left one appeared absolutely fibrotic. In our sections, we could not find a tubule or any sign of spermatogenesis. The right testicle was only three quarters destroyed; there were some persistent tubules and some mitotic figures among the spermatogonia. There was some effort at sperm formation, but it was incomplete and no sperm was present. I could not find any unmistakable interstitial cells in either testis. So that I think there is no doubt that there was extensive testicular destruction by mumps but, as one would expect, not complete destruction, and there must have been another reason for the marked depression in function of the remaining testicular tissue. The adrenal glands seemed a little small and the cortices possibly narrow, but histologically I could not see anything definitely abnormal. The thyroid gland was distinctly atrophied and weighed only 10 gm. Microscopically, the acini were small, and there was considerable increase in fibrous tissue. The pituitary gland was largely replaced by a neutrophilic, presumably nonfunctioning adenoma. Only a small rim of anterior lobe tissue persisted on one margin. It seems fairly evident that pituitary function was depressed because of compression by the tumor.

CASE 27292

Presentation of Case

A fifty-seven-year-old housewife entered the hospital complaining of pain in her leg.

The patient had been known to have hypertension for about four years, with systolic blood-pressure readings reaching as high as 230. She felt well, however, until four hours before admission, when a smothering sensation developed under the upper sternum, without dyspnea, pain or cough. She lay down, and half an hour later a gradually increasing pain and tingling appeared in her left leg and was soon followed by numbness, cold and paralysis of this extremity. There was also transient tingling in the right foot. Her physician gave her morphine subcutaneously.

The patient had complained of frequent head-

aches and slight dyspnea on exertion for the previous month. There was no history of rheumatic fever, chills, night sweats, anginal pain or substernal oppression.

The patient's mother had died of "shock"; one sister had hypertension.

On examination, the patient was pale and appeared anxious and acutely ill. The heart was enlarged to the left, with systolic and diastolic murmurs at the base, heard best over the aortic area. One examiner believed there might have been a mitral systolic murmur. The rhythm was regular. The blood pressure was 160 systolic, 110 diastolic. Occasional moist rales were heard throughout the chest. The abdomen was normal. The patient was unable to move her left leg, which was cold and a mottled pink below a definite line drawn horizontally through the lower margin of the vulva. In this leg no pulsations of the femoral, popliteal, posterior tibial and dorsalis-pedis arteries were palpable, and the sphygmomanometer did not oscillate below the mid-thigh or in the calf. Pulsations were present throughout the right leg.

The temperature was 99°F., the pulse 100, and the respirations 28.

An electrocardiogram showed normal P waves and QRS complexes in all four leads. The T waves were upright, but of low amplitude; the ST segments were very slightly depressed.

Two and a half hours after admission, the patient could move her left ankle, but not the toes. The leg was warmer, pulsations were palpable in the femoral artery to a point just below the profunda femoris branch, and the sphygmomanometer oscillated at the mid-thigh and calf, but not at the ankle.

Three hours after admission, an incision was made along the left femoral artery below Poupart's ligament under local anesthesia. Death occurred nine hours after admission.

Differential Diagnosis

Dr. Claude E. Welch: This is a short story, and it might be just as well to list first the few diagnoses that might be considered. These are peripheral embolus, a coronary attack, with marked spasm in the extremity, and a dissecting aneurysm of the aorta. Several points are of considerable importance in making the diagnosis. Of course, it is quite obvious as one reads the history that the clinical diagnosis was an embolus in the left leg.

I shall consider some features not consistent with that diagnosis. In the first place, the patient had had a hypertension for many years, but four hours before admission a smothering sensation in the chest developed. This is a very important symptom, it seems to me, because the patient remembered it well enough to tell about it. It could have been due either to dissecting aneurysm or to coronary thrombosis. It is of interest that we recently had under treatment a man with acute coronary thrombosis whose initial symptom was pain in the leg. This pain was so severe that it was not until several hours later, after he had been given morphine, that he recognized that he had pain in the chest. The pain in the leg turned out to be due to spasm, and it was later proved that he had an acute coronary thrombosis. A smothering sensation is not at all typical of embolus.

There is nothing in the past history of any interest.

Clinical examination of the heart would mean something to Dr. Bland, but it does not mean much to me. The electrocardiograms are of some interest because the T waves were upright. All I know about electrocardiograms is that with coronary disease the T waves are often inverted. This electrocardiogram therefore offers a little evidence against coronary disease. At the onset the findings in the leg were typical of occlusion at the level of the bifurcation of the iliac artery. Apparently, the patient's pulsations returned, in part, while she was on the ward. That would lead the surgeon to believe that the embolus had descended to a spot where it might be accessible.

Apparently operation was performed; at least the patient was explored for an embolus and died nine hours after admission.

If the symptoms had been due to embolus, there was no reason to expect death at this time. Patients die usually of an unrelated cause, — coronary thrombosis or gangrene, — days or even weeks later, so that death at that time is not consistent with the single diagnosis of embolus. It seems to me, therefore, that many features in this case are inconsistent with the diagnosis of embolus; some are inconsistent with coronary thrombosis. There is nothing inconsistent with the diagnosis of dissecting aneurysm of the aorta, and that is the one that I make.

Dr. Wyman Richardson: I should like to mention just one thing. I certainly agree with Dr. Welch, but I wonder if this is going to turn out to be the type of dissecting aneurysm that is due to media necrosis cystica, or whether syphilis may not actually have been present in this case.

I should like to ask Dr. Welch about spasm in connection with coronary disease. Is there a complete loss of pulsation?

DR. WELCH: In the case I mentioned, the patient had previously recognized vascular disease in the leg. There were no pulsations before or after the attack of coronary disease. We had another patient with absent pulsations immediately after an attack of coronary thrombosis; they returned after the acute phase had subsided.

DR. WILLIAM B. BREED: It seems obvious that the people who had charge of this patient did not think she had a dissecting aneurysm. They operated on the leg on the basis that she had an embolus. I am surprised that she did not have more pain in the chest, in the back and in the abdomen. Also, the fact that this happened rather suddenly makes me think that perhaps she did not have a dissecting aneurysm. I should favor an embolus, which was the diagnosis that the person who was in charge of her made.

DR. EDWARD F. BLAND: Is Dr. Welch disturbed by the diagnosis of dissecting aneurysm and by the finding of such good pulsations in the right leg in contrast with complete absence at first in the left, followed later by the appearance of pulsations in the upper portion of the affected leg? Do you often see that situation in dissecting aneurysm?

DR. WELCH: The peripheral circulation after dissecting aneurysms that go far down the aorta is very confusing. I am sure I have seen them follow a course like this in one or two cases. One has to assume a certain amount of spasm of the large vessels to account for the changing symptoms.

DR. BREED: The patient might perfectly well have died of a pulmonary embolus from the right side of the heart.

DR. BENJAMIN CASTLEMAN: That is very rare.

DR. BREED: Pulmonary emboli associated with coronary disease or occlusion are not at all uncommon.

DR. CASTLEMAN: The source of the emboli is very rarely from a mural thrombus overlying a right ventricular infarct. They may arise from an auricular thrombus associated with fibrillation or from the peripheral veins in the legs.

DR. RICHARDSON: I think the patient died of dissecting aneurysm, probably of pericardial tamponade.

DR. WELCH: Would it be amiss to ask Dr. Bland to interpret the cardiac situation for us?

DR. BLAND: The electrocardiogram is all right from the description. The diastolic murmur as described means aortic regurgitation. We do not know about the blood serologic findings, of course. An aortic diastolic murmur appears after dissection in a number of cases, probably because of distortion of the aortic valve ring, owing to dissection backward toward the heart. If this murmur had not been present before and had appeared after the episode, it would be strong evidence in favor of dissection of the aorta.

DR. BREED: On the other hand, the electrocardiogram was taken only two hours after the onset of the disease, and you certainly could not rule out coronary disease or occlusion by an electrocardiogram taken at that time, could you?

DR. BLAND: No.

DR. CASTLEMAN: The surgeon is not here, but I shall read his operative note:

> A careful re-examination of the extremity was made just before operation, to be sure that the signs were not improving as they had previously. The level of demarcation was the same, — just below the knee, — the foot was cold, the popliteal pulse was still absent, and the femoral pulsation was the same as previously.
>
> A 10-cm. longitudinal incision was made under novocain, extending from Poupart's ligament downward over the course of the femoral artery. The artery was found to be pulsating throughout the entire extent of the exposure, which was for 4 cm. below the origin of the profunda femoris. The profunda itself was also pulsating, although all pulsations were definitely diminished. There was apparently some inflammatory reaction around the artery, since the femoral vein was quite adherent to it. The vein was dissected off and divided, and it was found that the upper end of the vein bled profusely, showing that there was no thrombosis in it.
>
> Attention was then turned to the artery. After some deliberation, and in view of the definite clinical findings, it was decided to open the artery; this was done longitudinally through a 1-cm. incision, 4 cm. above the profunda. A free flow was obtained from above, moderately free flow from below. No thromboses or emboli were seen or extractable. The artery was then closed.

CLINICAL DIAGNOSES

Embolus to left femoral artery.
Hypertensive heart disease.

DR. WELCH'S DIAGNOSIS

Dissecting aneurysm of aorta.

ANATOMICAL DIAGNOSIS

Dissecting aneurysm of aorta.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: At autopsy, we found an enlarged heart, weighing 700 gm. The coronary arteries showed a moderate degree of sclerosis but no evidence of occlusion. In the ascending aorta, several centimeters above the valve, was a T-shaped intimal tear in the intima, which communicated with a dissecting aneurysm. The ex-

ternal perforation had occurred just below this region into the pericardium, which was full of fresh blood. The dissection had continued down the aorta into the left external iliac and femoral arteries, to a point about 5 cm. below Poupart's ligament, at which point the lumen was occluded by the opposing intimal surfaces. The incision at operation was a few centimeters below the point of occlusion. Had the surgeon known that this was a dissecting aneurysm at the time of operation, he might have attempted the operation described by Gurin,* that is, the making of a nick in the occluding intima above, to allow the blood from the dissecting channel to come down and enter the original stream. This might have relieved the pressure in the aneurysm and perhaps prevented external rupture. In Gurin's case, the circulation of the leg was restored, but the patient died six days later.

There is one other thing that occurred in this case that is quite unusual. The external perforation through the adventitia must have been relatively slow, because the blood had extended from the adventitia of the aorta into the adventitia of the main pulmonary artery and its branches into both lungs, appearing as subpleural hemorrhagic extravasations in the interlobar septa. At present, we have not finished studying the microscopic sections, but we are quite certain that the etiology of the dissection was media necrosis aortica cystica. There were no atheromatous plaques or areas of arteriosclerosis in the ascending aorta at the site of the tear. There was no evidence of syphilis or of an inflammatory reaction that would go with syphilis. I might say, Dr. Richardson, that syphilis rarely, if ever, produces dissecting aneurysm.

*Gurin, D., Bulmer, J. W., and Derby, R. Dissecting aneurysm: diagnosis and operative relief of acute arterial obstruction due to this cause. *New York State J. Med.* 35:1200-1202, 1935.

Dr. Richardson: My reasoning was false. I did not realize that a diastolic murmur could occur as a result of a dissecting aneurysm. If I had known this, I should not have mentioned syphilis.

Dr. Bland: Was there any abnormality of the aortic valve, other than the deformity of the aortic ring, to account for the diastolic murmur?

Dr. Castleman: No.

Dr. Bland: Dr. Chester S. Keefer once suggested that such a murmur might be due to the reflux of blood through the rent in the aorta, a possibility that seems to me most unlikely. I do not believe there is that much reflux of blood with diastole, unless possibly at a much later date when a double aorta has developed in the few who survive. The first explanation seems more reasonable. In perhaps a fourth of the cases, an aortic diastolic murmur appears immediately after the dissection.

Does syphilis of the aorta protect against this condition?

Dr. Castleman: I do not know. It is quite possible that the medial scars that are formed in syphilis might be a protection against it. We have never seen dissecting aneurysms of the aorta produced by syphilis.

Dr. Breed: May I ask again about the pain in dissecting aneurysm? Do you think the pain in this case was less? I am not trying to defend my diagnosis. I am asking for information.

Dr. Bland: I should say that at least 90 per cent of patients have severe pain. Double-barreled aortas are occasionally found post mortem, with no history of an acute illness corresponding to the dissection.

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POPULARIZATION OF ADVANCES IN MEDICINE

The June 14 issue of *Science News Letter*, a weekly publication devoted to popularized accounts of advances in the sciences, makes the following startling introductory statement concerning papers presented at the Cleveland meeting of the American Medical Association: "*Six new successful treatments, promising relief or cure for the suffering of thousands of men and women, were reported. . . . Hundred per cent control of gonorrhea is made possible by sulfa drug. Crude liver extract is the new remedy for gout and arthritis. Salts of gold are being tried secretly on human rheumatism patients. Sex hormone treatment gives new hope for those with cancer of prostate gland. Iodized oil helps sinus disease, and x-rays are advised for Hodgkin's disease.*" As one reads the subsequent paragraphs, one is told that the drug, sulfathiazole, has been responsible for 100 per cent cures of gonorrhea in three days; that the use of crude liver extract in the treatment of patients with gout and arthritis was promptly followed by a disappearance of symptoms, particularly in the former disease; that a gold compound, calcium aurothiomalate, has been successfully used in the treatment of experimentally produced arthritis in mice; that female sex hormones or castration inhibits the growth of cancer of the prostate; that the injections of iodized oil into the sinuses result in the subjective improvement of patients suffering from sinusitis; and that patients with Hodgkin's disease are relieved by x-ray or radium treatment.

In commenting on these abstracts, the medically trained writer could reasonably state that sulfathiazole is a standard accepted remedy for gonorrhea, although no one has ever before obtained 100 per cent effectiveness, a claim that requires careful follow-up studies over a period of months or years; that in judging the value of any new treatment for gout, — the present treatment is reasonably satisfactory, — the possibility of spontaneous remissions of the disease must be excluded; that experimentally produced arthritis in mice is undoubtedly a different disease from human arthritis, the cause of which is unknown; that the efficacy of the use of female sex hormones in the treatment of cancer of the prostate has been discussed, pro and con, for several years; that subjective improvement in patients with sinusitis is a poor yardstick for measuring the value of any therapeutic agent; and that x-rays and radium have been used for the treatment of patients with Hodgkin's disease for several decades. In other words, three of these "new, successful treatments" have been previously reported, two require confirmation of their value, and for one no claim for efficacy in human beings has been made. Undoubtedly many, if not all, of these points were brought out by the speakers at the meeting, and it is unfortunate that popularized reports in periodicals and newspapers are, in many instances, so garbled.

The public should, of course, be informed concerning advances in medicine, since lay education

of this sort is one, if not the most effective, means of promoting the health of the nation. However, such reports, particularly those in regard to widespread diseases such as gonorrhea, arthritis and sinusitis, should be accurate and conservative; if "successful remedies" are announced year after year, the layman will become suspicious, and lay support of the medical profession — a matter of vital importance at the moment — will be weakened.

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MEDICAL EXPLORERS

Not a few physicians who have traveled extensively have returned home to write of their explorations and thus to give pleasure and instruction to their fellow colleagues and many others. Some have set out on scientific missions, such as Mungo Park, the young Scottish surgeon, whose *Travels in the Interior Districts of Africa* (1799) may still be read as a thrilling account of individual adventure. Other famous doctors described their travels for pleasure. Two may be mentioned: Richard Bright's *Travels from Vienna through Lower Hungary* (1818), an account of a postgraduate tour, illustrated with Bright's original drawings, and Thomas Hodgkin's *Narrative of a Journey to Morocco* (1866), the story of a trip with his patient, Sir Moses Montefiore. To these two groups must be added a third, composed of those describing a vacation tour or a particular service with the Army or Navy. In this category would fall the well-known book by Oliver Wendell Holmes, *Our Hundred Days in Europe* (1887), and the fine account by the late Harvey Cushing entitled *From a Surgeon's Journal* (1936).

The whole group of travel books by doctors extends into several thousand volumes. There have already been gathered in one library four or five hundred volumes, and many more are known. Doctors in general have added greatly to the knowledge of the world, and their reminiscences have been of much interest to their fellow colleagues. Few, however, have engaged in much more than the ordinary types of travel. Mungo Park would be considered an explorer of the first class, as would the American, Elisha Kent Kane, who visited the Arctic.

At the present time there must be added to these individuals the name of Richard Upjohn Light, who in the last ten years has made two unusual trips by aeroplane, accounts of which have been published. The first one was a privately printed book, *Journal of a Seaplane Cruise Around the World, August 20, 1934, to January 24, 1935* (1937). On this trip, Light, with Robert French Wilson as his radio operator, circumvented the globe by aeroplane, except for the trip across the Pacific from Manila to Vancouver. Thus he became the first American to fly from continental United States eastward to the Philippine Islands. His log of the journey was accompanied by his carefully drawn maps of the country over which he passed. He made many notes of particular medical interest, and one of his reports, after his visit to Labrador, was separately published in the October 25, 1934, issue of the *Journal*. In addition there are many notes in the book about medical conditions in Greenland and accounts of his visits to various hospitals in Europe. This journey, taken seven years ago, was a pioneer effort in exploration by aeroplane and was widely recognized by geographers as a trip of unusual importance. Following this exploration, Light was made a member of the American Geographical Society and later was elected a councilor of that distinguished scientific body.

Under the auspices of the American Geographical Society, Light has now published his account of another aeroplane trip, this time over Africa. A review of the extraordinary book appears in this issue of the *Journal*. His account is a fitting climax to an unusual career in the air, which began in 1930. Light flew over Central America in 1932: later he photographed parts of the Rocky Mountains from Vera Cruz in Mexico northward to Salt Lake City. His trip around the world in 1934–1935 has already been mentioned. In 1937–1938, prior to the trip across Africa, Light and his wife flew westward across North America, southward through Mexico and Central America, then eastward across South America to Rio de Janeiro. During this decade, Light has become the outstanding physician of his time in the world of aerial exploration.

MEDICAL EPONYM

Hutchinsonian Teeth

On May 18, 1858, Jonathan Hutchinson (1828–1913) presented "Report on the Effects of Infantile Syphilis in Marring the Development of the Teeth" before the Pathological Society of London. This paper appears in the *Transactions of the Pathological Society of London* (**9:** 449-455, 1858).

> For a considerable time past, I have been in the habit of recognising in a certain very peculiar development of the permanent teeth an indication that their possessor had in infancy suffered from hereditary syphilis. . . . I will now pass to the conclusions arrived at.
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> . . . . *There is a peculiar condition of the teeth, which results from the influence of hereditary syphilis, and . . . the most frequent features of this condition are the following*: — *a. Smallness.* — The teeth stand apart with interspaces, and are rounded and peggy in form instead of flat. *b. Notching.* — They usually exhibit in their border a broad shallow notch, or at times, two or three (serrated). Owing to their softness, these teeth rapidly wear away, and this notching is thus often obliterated, but when markedly present, it is one of the most decisive conditions. *d. Colour.* — Instead of the clear, smooth, white exterior of good teeth, they present a dirty greyish surface, totally destitute of polish and rarely smooth. No amount of cleaning will materially alter this feature which owes its existence, I believe to the great deficiency of enamel. *e. Wearing down.* As before observed their softness from deficiency of enamel renders them liable to premature wearing down. . . . f. The signs mentioned apply almost exclusively to the incisors and canines and in fact the grinders are usually altered in a very much less degree. Their surfaces are often more uneven than those of healthy teeth, and now and then they present tubercular projections of a very peculiar character. . . . The above remarks apply only to the permanent set.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Sudden Death of Unknown Cause During the Sixth Month of Pregnancy

A twenty-seven-year-old para II had had no prenatal care. Physical examination during her first pregnancy, two years previously, was negative; the blood pressure and the heart and lungs were normal. There was no history of operation. The first pregnancy had terminated normally at term with a living child. The physician who had attended the patient previously was informed of the present pregnancy very casually when making a visit at the house because the child had measles. He made no examination. A very short time after this, the patient complained of a severe pain and died before a physician arrived.

Comment. Since it is known that emboli during pregnancy cause death, it seems likely that such a condition was the cause of sudden death in this case. There was no history of heart disease. The patient had had a baby two years earlier, at which time the attendant physician said that her heart was normal. She had had no infections since then, so that it is fair to infer that there was no cardiac lesion. The blood pressure was said to have been normal with her first pregnancy, and consequently hypertension as a cause of cerebral hemorrhage can be ruled out. Furthermore, had she had a cerebral hemorrhage, it is unlikely that she would have died so quickly. It is said that she had a pain and died. It is not stated where the pain was localized. The uterus may rupture spontaneously at five or six months, but death occurring so quickly after the rupture is unlikely, and there was no history of vaginal bleeding.

It is unfortunate that the medical examiner did not insist that an autopsy be performed.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

DEATHS

DOUGHERTY — Harry L. Dougherty, M.D., of Jamaica Plain, died June 1. He was in his forty-third year.

Born in Nickelsville, Virginia, he attended William and Mary College and received his degree from the University of Louisville School of Medicine in 1925. He served his internship at the United States Marine Hospital in Chelsea and then opened offices in Jamaica Plain. He was a member of the staff of the Faulkner Hospital.

Dr. Dougherty was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, his parents, two daughters, two sons, three brothers and four sisters survive him.

NORTON — George P. Norton, M.D., of Fitchburg, died June 12. He was in his sixty-eighth year.

Dr. Norton received his degree from the Bellevue Hospital Medical College, New York City, in 1895. He was former city physician and district medical examiner and had been on the staff of the Burbank Hospital for twenty-nine years. At the time of his death, he was chief medical examiner for the draft board under the Selective Service Act.

A former president of the Worcester North District Medical Society, he was also a member of the American Medical Association.

His widow, two sons, a daughter and a grandchild survive him.

TRACY — JOHN M. TRACY, M.D., of Springfield, died July 9. He was in his seventy-fourth year.

Born in Saugerties, New York, he attended St. Mary's College, New York City, and received his degree from George Washington University School of Medicine in 1898.

Dr. Tracy started practice in Springfield in 1899, where he was a former member of the School Committee and a member of the staff of the Mercy Hospital and the city health department. At the time of his death, he was a lieutenant colonel in the United States Army Medical Reserve.

He was a fellow of the Massachusetts Medical Society and the American Medical Association.

Two sons and a daughter survive him.

MISCELLANY

GASTRIC LAVAGE IN ADULTS

Failure to find tubercle bacilli in the sputum, even after repeated examination of successive specimens, is no justification for ruling out the possibility of tuberculosis. The least certain of the methods commonly used is the staining of the direct smear of the untreated sputum. One supplementary method is to examine the stomach washings for tubercle bacilli, especially in children, who are likely to swallow the sputum. Roper and Ordway (Gastric Lavage in Adults with Pulmonary Tuberculosis, *Am. Rev. Tuberc.*, 43:543–556, 1941) advocate its wider use for adults and offer impressive evidence of its value. Abstracts of their article follow:

The examination of fasting gastric contents by smears for tubercle bacilli, first reported in 1898, proved unreliable, and the need for culture and animal inoculation was demonstrated. Since 1927, numerous articles have been published regarding this procedure in juvenile tuberculous patients. More recently the test has been used on adults.

This report presents the findings obtained by guinea-pig inoculations of fasting gastric contents of tuberculous and nontuberculous patients. By the addition of this procedure to the usual methods, recovery of tubercle bacilli in tuberculous patients was practically doubled.

The studies were carried out at the Metropolitan Life Insurance Company Tuberculosis Sanatorium during a three-year period in which approximately 1000 patients were admitted, of whom 135 were diagnosed as having active pulmonary tuberculosis and the remainder were considered to be nontuberculous. This afforded opportunity for using controls.

Since 1929, the percentage of minimal cases admitted has almost trebled; in patients with moderately advanced disease it has decreased slightly, and in those with far advanced involvement it has declined to one third the former figure. Yet the percentage of positive sputum cases has fallen despite the more frequent use of animal inoculation (from 45.2 per cent in the period 1926 to 1928 to 34.8 per cent in the period May, 1936, to May, 1939).

Of the 135 tuberculous patients of the present study, 34.8 per cent gave positive recovery by sputum examinations alone. The addition of gastric lavage almost doubled this percentage, namely, 63 per cent.

Sharp distinction is made between sputum and fasting stomach contents. Sputum refers to the bronchial secretion that is actually expectorated by coughing or clearing the throat. The gastric specimen contains that bronchial secretion which has gained entrance into the pharynx and has subsequently been swallowed. Gastric lavage was initially employed in children because of their inability to expectorate. By means of this same test in adults, many positive results are obtained among those whose efforts to raise sputum are unsuccessful, as well as in many of those producing unsatisfactory or negative sputum.

Sputum produced by the tuberculous patient may contain tubercle bacilli on one day and none on the next. The same variability occurs with gastric washings. Stiehm recommends that the test be given on each of three successive mornings.

The test is of assistance not only in the diagnosis but also in the management of the tuberculous patient. After years of treatment, the sputum may disappear or become negative, whereas the gastric contents may still exhibit virulent tubercle bacilli.

The procedure of obtaining the gastric specimen causes only slight discomfort to most persons and is not harmful; on the other hand, repeated forceful voluntary efforts to expectorate are uncomfortable and may be harmful. The value of proving or disproving the clinical and roentgenologic diagnosis of pulmonary tuberculosis is obvious; and of equal significance is the conclusive demonstration of the subsequent disappearance of tubercle bacilli from the bronchial secretions of tuberculous patients under treatment. Knowledge of these facts is of sufficient import to justify the use of gastric lavage whenever it is indicated by the absence or negativity of sputum. — Reprinted from *Tuberculosis Abstracts*, July, 1941.

CORRESPONDENCE

"THE CLOSED-STAFF HOSPITAL"

To the Editor: An editorial in the June 5 issue of the *Journal* pointed out many of the advantages of the closed-staff hospital but failed to mention any of the unsalutary practices that sometimes exist.

That any member of the medical profession who is "technically equipped, adequately trained, ethical and desirous of keeping constantly up to date in his profession will have no difficulty in obtaining permission to practice in his local hospital" is not always true. Members of medical boards who are responsible for nominations to the staff are not always actuated by purely altruistic motives. Prejudice, jealousy and ambition to increase their practice regardless of the Golden Rule may influence their decisions. Moreover, members of these committees are often self-perpetuating either by reason of their position as chiefs of services or because of their appointment by lay boards who govern the hospital. Nominations to the staff, therefore, are not always based on the "considered judgment of the doctors' peers," since they are made by physicians who may not be responsible to or representative of the staff. As a result, members of medical boards may prohibit a physician from using the hospital for an indefinite time without just cause; this interferes with his livelihood, prevents his professional development and results in inferior service to his patients.

The correction of these unfair practices is definitely the business of organized medicine, such as the Massachusetts Medical Society. If physicians who serve on executive or advisory committees in hospitals could be elected by ballot by the entire staff for limited terms (possibly one to three years), it would not only reduce these discriminatory practices but also contribute to the democratic administration of hospitals as a whole. Such recommendation by the Massachusetts Medical Society would no doubt be received by hospital trustees or directors with

the same spirit of co-operation as were similar constructive suggestions by other reputable medical organizations, such as the American Medical Association and the American College of Surgeons.

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BOOK REVIEWS

Focus on Africa. By Richard Upjohn Light, M.D. Foreword by Isaiah Bowman, Ph.D., LL.D. 4°, cloth, 228 pp., with 323 illustrations and 14 maps. New York: American Geographical Society, 1941. $5.00.

In the winter of 1937–1938, Dr. Richard Light, an explorer of note, who has traveled extensively in his aeroplane, and also a neurosurgeon, now in practice in Kalamazoo, Michigan, who was trained by Dr. Harvey Cushing, flew across Africa from Capetown to Cairo. His wife, who is also an expert in aviation, acted as co-pilot, radioman and photographer. This extraordinary trip is now described in a narrative augmented by a series of superb photographs of the country over which he flew. Dr. Light writes in a delightful and informative style, and the book will make delightful reading for physicians interested in the broader aspects of medicine and travel.

Although there is not much strictly medical material in Dr. Light's account, nevertheless he gives sidelights on the people of South Africa and some of the problems connected with the immense industries of gold and diamond mining. In addition to the unusual photographs giving a new idea of the terrain of a country as viewed from the air, the book is illustrated by numerous maps carefully depicting the course of his flight. It may well be said, as noted by Dr. Isaiah Bowman, who writes a foreword to the book, that Dr. Light has put details "against their parent backgrounds, effects against their causes" in an admirable and graceful manner. He is indeed one of the leaders of modern exploration whose standard is "To broaden those earth-bound glimpses. . . ."

Lipidoses: Diseases of the cellular lipid metabolism. By Siegfried J. Thannhauser, M.D., Ph.D. Edited by Henry A. Christian, M.D., LL.D., Sc.D. (Hon.), F.R.C.P. (Hon., Can.). (Reprinted from *Oxford Loose-Leaf Medicine.*) 8°, cloth, 370 pp., with 78 illustrations. New York: Oxford University Press, 1940. $6.00.

Harvey Cushing once remarked that the field of general surgery at first appealed to him as being most worth while to master; before long he realized that surgery of the central nervous system was less well understood and therefore was more appealing. This quickly proved to be too broad an area for his temperament, and hence surgery of the brain became of paramount importance. But the brain was too large so that next its center, the pituitary gland, attracted his main attention; finally the secrets of the anterior lobe of this small body proved sufficiently elusive to satisfy his medical inquisitiveness. This is a good story of how zeal to make important discoveries in a small corner of medicine is often developed.

The author of this book evidently feels about medical research as Cushing did. For he began as a competently trained teacher and investigator of general medicine: his imagination became stimulated by how little was known of the general subject of the lipids, and this book is the crystallization of twenty years of painstaking, critical study of the matter.

The Oxford University Press always publishes stylishly. Hence the format of the volume is excellent. The type is easy and agreeable to read, the illustrations on the whole are clear, and the job of indexing is well done. The plan of the work, too, is easily comprehensible: first a discussion of the physiology and chemistry of lipid metabolism; next a chapter on hyperlipemia; then chapters on the xanthomatoses, primary, secondary or localized; and finally two concluding chapters on those confusing diseases known familiarly to the well read as Gaucher's disease and Niemann–Pick's disease. A scholarly bibliography of some eight hundred references is neatly dovetailed in between each chapter. The net result is a fine monograph that will serve for the next several years as the standard work of reference about unique disorders caused by disturbances in the metabolism of fat.

Photodynamic Action and Diseases Caused by Light. By Harold F. Blum, Ph.D. 8°, cloth, 309 pp., with 25 tables and 50 illustrations. New York: Reinhold Publishing Corporation, 1941. $6.00.

This book justifies the two distinct purposes that prompted the American Chemical Society to sponsor a series of scientific and technologic monographs on chemical subjects. First, it closely connects the fields of physics, chemistry and medicine in the study of photodynamic action and diseases caused by light. Secondly, it presents a thorough historical survey of the progress already made, and points out directions in which research should be extended.

This study is vastly important at a time when industry is introducing many new chemicals, especially dyes, in our defense program. Their possible toxicity in relation to light has been recently described. This deleterious combination has been recognized for almost the past five decades, but has been neglected by most physicians. The author's attempts to correct or clear many incorrect or vague statements in the literature are worth while. Many diseases have been ascribed without proof to the action of sunlight. Other diseases are readily recognized in certain endemic areas, but are missed in other places because of ignorance of their existence. Few physicians recognize this sensitivity to light among human beings. Today, with the widespread use of ultraviolet radiation and the increase in the use of photodynamic substances for diagnostic purposes and in chemotherapy, physicians should be alert and cognizant of their possible effects when the patient is subjected to sunlight, to radiation from mercury-vapor lamps and carbon-arc lamps and to roentgen rays, for great damage can be done. Dermatologists have been more or less aware of the untoward results that occur after the unfortunate use of these substances without knowledge of them. This has been demonstrated in the use of dyes and ultraviolet radiation in the treatment of psoriasis, and their use in cosmetics and industrial chemistry.

The book is divided into four parts: the introduction describes the nature of radiation and its general biologic effects; the second part presents the general photochemical aspects of photodynamic action; the third section describes the diseases caused by light in domestic animals; and the fourth part describes the diseases produced by light in man. Each phase is adequately treated, and each part can be read independently.

It is a book that can be found useful by all students, and it should be in the library of all physicians and veterinarians.